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=> d his

     (FILE 'HOME' ENTERED AT 08:12:09 ON 04 MAY 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 08:12:39 ON 04 MAY 2004
L1      2087394 S CALCIUM OR CALMODULIN
L2        38966 S L1 (2W)KINASE?
L3         8783 S HUMAN AND L2
L4      6504693 S CLON? OR EXPRESS? OR RECOMBINANT
L5         4552 S L3 AND L4
L6      4062032 S BRAIN OR LUNG OR HIPPCAMPUS
L7          771 S L5 AND L6
L8           78 S HUMAN(W)L2
L9           17 S L7 AND L8
L10           8 DUP REM L9 (9 DUPLICATES REMOVED)
                E YAN C/AU
L11         996 S E3
                E KETCHUM K/AU
L12         193 S E7-E9
                E MERKULOV G/AU
L13          79 S E7-E9
                E BEASLEY E M/AU
L14         291 S E3
L15        1449 S L11 OR L12 OR L13 OR L14
L16           5 S L3 AND L15
L17           4 DUP REM L16 (1 DUPLICATE REMOVED)
                SET NOTICE DISPLAY 1

     INDEX 'IFICLS, PATOSEP, PATDPA, INPADOC' ENTERED AT 08:25:38 ON 04 MAY
     2004
                SEA US 6387677/PN,APPS
               ---------
               1    FILE IFICLS
               1    FILE INPADOC
L18             QUE US 6387677/PN,APPS
               ---------

     FILE 'IFICLS, INPADOC' ENTERED AT 08:25:41 ON 04 MAY 2004
L19             2 S L18
                SET NOTICE LOGIN DISPLAY

     FILE 'MEDLINE, EMBASE, BIOSIS, SCISEARCH, HCAPLUS, LIFESCI' ENTERED AT
     08:28:57 ON 04 MAY 2004
L20            68 DUP REM L8 (10 DUPLICATES REMOVED)

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 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  JAN 27   Source of Registration (SR) information in REGISTRY updated
                   and searchable
 NEWS  4  JAN 27   A new search aid, the Company Name Thesaurus, available in
                   CA/CAplus
 NEWS  5  FEB 05   German (DE) application and patent publication number format
                   changes
 NEWS  6  MAR 03   MEDLINE and LMEDLINE reloaded
 NEWS  7  MAR 03   MEDLINE file segment of TOXCENTER reloaded
 NEWS  8  MAR 03   FRANCEPAT now available on STN
 NEWS  9  MAR 29   Pharmaceutical Substances (PS) now available on STN
 NEWS 10  MAR 29   WPIFV now available on STN
 NEWS 11  MAR 29   No connect hour charges in WPIFV until May 1, 2004
 NEWS 12  MAR 29   New monthly current-awareness alert (SDI) frequency in RAPRA
 NEWS 13  APR 26   PROMT: New display field available
 NEWS 14  APR 26   IFIPAT/IFIUDB/IFICDB: New super search and display field
                   available
 NEWS 15  APR 26   LITALERT now available on STN
 NEWS 16  APR 27   NLDB: New search and display fields available

 NEWS EXPRESS  MARCH 31 CURRENT WINDOWS VERSION IS V7.00A, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
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FILE 'HOME' ENTERED AT 08:12:09 ON 04 MAY 2004

=> file medline embase biosis biotechds scisearch hcaplus ntis lifesci
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21
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FILE 'LIFESCI' ENTERED AT 08:12:39 ON 04 MAY 2004
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=> s calcium or calmodulin
L1     2087394 CALCIUM OR CALMODULIN

=> s l1 (2w)kinase?
L2       38966 L1 (2W) KINASE?

=> s human and l2
L3        8783 HUMAN AND L2

=> 's clon? or express? or recombinant
'S IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s clon? or express? or recombinant
   5 FILES SEARCHED...
L4     6504693 CLON? OR EXPRESS? OR RECOMBINANT

=> l3 same l4
L3 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l3 and l4
L5        4552 L3 AND L4

=> s brain or lung or hippcampus
L6     4062032 BRAIN OR LUNG OR HIPPCAMPUS

=> s l5 and l6
L7         771 L5 AND L6

=> s human(w)l2
L8          78 HUMAN(W) L2
```

=> s l7 and l8
L9 17 L7 AND L8

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 8 DUP REM L9 (9 DUPLICATES REMOVED)

=> d 1-8 ibib ab

L10 ANSWER 1 OF 8 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2004091023 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14980499
TITLE: Effects of PU.1-induced mouse **calcium-calmodulin**-dependent **kinase** I-like kinase (CKLiK) on apoptosis of murine erythroleukemia cells.
AUTHOR: Yamada Toshiyuki; Suzuki Mitsuhiro; Satoh Hitoshi; Kihara-Negishi Fumiko; Nakano Hiroyasu; Oikawa Tsuneyuki
CORPORATE SOURCE: Department of Cell Genetics, Sasaki Institute, Tokyo 101-0062, Japan.. oikawa@sasaki.or.jp
SOURCE: Experimental cell research, (2004 Mar 10) 294 (1) 39-50. Journal code: 0373226. ISSN: 0014-4827.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200403
ENTRY DATE: Entered STN: 20040225
Last Updated on STN: 20040331
Entered Medline: 20040330

AB PU.1, a hematopoietic cell-specific Ets family transcription factor, is involved in the generation of murine erythroleukemia (MEL). To identify the target gene(s) of PU.1 in MEL cells, we carried out differential display (DD) analysis and isolated a novel gene whose **expression** was up-regulated after overexpression of PU.1 in MEL cells. Because the gene exhibited about 90% homology with the **human calcium-calmodulin**-dependent **kinase** I-like kinase (CKLiK) gene, it was identified as a mouse homologue of **human** CKLiK. The mCKLiK gene was mapped to the mouse chromosome 2A1-A3 region and shown to be **expressed** predominantly in T cells lymphoma and embryonal carcinoma cell lines and primary thymus and **brain**. Two types of transcripts were present showing a difference in the 3' portion of the coding region and CREB-activating ability. Overexpression of each isoform of mCKLiK in MEL cells revealed that one of them induces, while the other inhibits apoptosis under low serum condition. Differentiation inhibition and lineage switch to myelomonocytes, which were previously observed in MEL cells overexpressing PU.1, were not provoked in the cells overexpressing mCKLiK. These results suggest that mCKLiK is up-regulated by PU.1 in MEL cells and involved in apoptosis of the cells.

L10 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:367221 HCAPLUS
DOCUMENT NUMBER: 136:364976
TITLE: Protein, gene and cDNA sequences of a novel **human calcium/calmodulin** dependent **kinase** sequence homolog
INVENTOR(S): Ye, Jane; Yan, Chunhua; Di Francesco, Valentina; Beasley, Ellen M.
PATENT ASSIGNEE(S): PE Corporation (NY), USA
SOURCE: U.S., 85 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6387677	B1	20020514	US 2001-800960	20010308
WO 2002088344	A1	20021107	WO 2002-US6686	20020305

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1373486	A1	20040102	EP 2002-733827	20020305

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

US 2002132325	A1	20020919	US 2002-96960	20020314
US 6664085	B2	20031216		

PRIORITY APPLN. INFO.: US 2001-800960 A 20010308
WO 2002-US6686 W 20020305

AB The invention provides protein, cDNA and genomic sequences for a novel **human** protein, which shares sequence homol. to a known **calcium/calmodulin** dependent **kinase**. The **calcium/calmodulin** dependent **kinase** sequence homolog gene is **expressed** in **human** placenta, breast (including mammary adenocarcinoma), skin melanotic melanoma, ovary adenocarcinoma, uterus leiomyosarcoma, Burkitt's lymphoma (lymph), duodenal adenocarcinoma (small intestine), and fetal **brain**. Sixteen novel single nucleotide polymorphism sites (beyond the ORF or in intron regions) have been identified on **calcium/calmodulin** dependent **kinase** sequence homolog gene. Thus, the present invention specifically provides isolated protein and nucleic acid mols., methods of identifying orthologs and paralogs of the **calcium/calmodulin** dependent **kinase** proteins, methods of identifying modulators of the **calcium/calmodulin** dependent **kinase** proteins, and methods of diagnosis and treatment of diseases associated with the **calcium/calmodulin** dependent **kinase**.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:886475 HCAPLUS
DOCUMENT NUMBER: 136:32798
TITLE: Protein, gene and cDNA sequences of **human** **calmodulin**-binding protein **kinase** sequence homolog
INVENTOR(S): Yan, Chunhua; Wei, Ming-hui; Ketchum, Karen; Merkulov, Gennady; Beasley, Ellen M.
PATENT ASSIGNEE(S): Applera Corporation, USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001092492	A2	20011206	WO 2001-US17327	20010530
WO 2001092492	A3	20020704		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2002015987	A1	20020207	US 2000-734030	20001212
US 6461846	B2	20021008		
EP 1290188	A2	20030312	EP 2001-948244	20010530
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003534794	T2	20031125	JP 2002-500684	20010530
US 2002142430	A1	20021003	US 2002-153921	20020524
US 6653116	B2	20031125		
US 2004038363	A1	20040226	US 2003-669689	20030925

PRIORITY APPLN. INFO.:

US 2000-207281P	P	20000530
US 2000-734030	A	20001212
WO 2001-US17327	W	20010530
US 2002-153921	A3	20020524

AB The invention provides protein and cDNA and genomic sequences for a novel **human** protein kinase, which has strong sequence homol. with rat, fish and Drosophila **calmodulin**-binding protein **kinases** . The gene is **expressed** in the fetal and adult **brain**, **lung**, and hippocampus. Nine single nucleotide polymorphism sites (beyond the ORF or in intron regions) were identified. Thus, the present invention specifically provides isolated peptide and nucleic acid mols., methods of identifying orthologs and paralogs of the protein kinase peptides, methods of identifying modulators of the protein kinase peptides, and methods of diagnosis and treatment of diseases associated with the protein kinase.

L10 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:	2001:812257 HCAPLUS
DOCUMENT NUMBER:	136:320118
TITLE:	Molecular **cloning** and sequence analyses of calcium/**calmodulin**-dependent protein **kinase** II from fetal and adult **human** **brain**. Sequence analyses of **human** **brain** calcium/**calmodulin**-dependent protein **kinase** II
AUTHOR(S):	Li, Guangyu; Laabich, Aicha; Liu, L. Olivia; Xue, Jin; Cooper, Nigel G. F.
CORPORATE SOURCE:	Department of Anatomical Sciences and Neurobiology, University of Louisville School of Medicine, Louisville, KY, 40202, USA
SOURCE:	Molecular Biology Reports (2001), 28(1), 35-41 CODEN: MLBRBU; ISSN: 0301-4851
PUBLISHER:	Kluwer Academic Publishers
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB The aims of this study were to characterize specific mRNAs and the **expression** pattern for isoforms of calcium/**calmodulin** -dependent protein **kinase** II (CaMKII) in the **human** **brain**. We **cloned** and sequenced the CaMKII α and β subunit cDNAs, and used them to study the CaMKII **expression** in **human** **brain**. Four distinct isoforms of CAMKII were isolated. Two of them were characterized as CaMKII α and β subunits. The other two showed similar nucleotide sequences, but one had a 33-bp insertion relative to the α subunit, and the other had a 75-bp deletion relative to the β subunit. These alterations are located within the variable regions. These two isoforms were characterized as CaMKII αB and βe. Northern blot anal. showed

that a 4.4-kb mRNA for the α isoform and a 3.9-kb mRNA for the β isoform were **expressed** in both **human** fetal and adult **brain** to different degrees. The results indicate that CaMKII **expression** is developmentally regulated. The CaMKII isoform **expression** was confirmed in **human** fetal and adult **brain** using RT-PCR with specific primers, which flanked the CaMKII variable regions. The CaMKII α, αB, β, β' and βe isoforms were characterized in both **human** fetal and adult **brain**.

REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1998:783640 HCAPLUS

DOCUMENT NUMBER: 130:150193

TITLE: Components of a **calmodulin**-dependent protein **kinase** cascade. Molecular **cloning**, functional characterization and cellular localization of Ca2+/**calmodulin**-dependent protein **kinase** kinase β

AUTHOR(S): Anderson, Kristin A.; Means, Raylene L.; Huang, Qi-Hui; Kemp, Bruce E.; Goldstein, Elaine G.; Selbert, Michele A.; Edelman, Arthur M.; Fremeau, Robert T.; Means, Anthony R.

CORPORATE SOURCE: Department of Pharmacology and Cancer Biology, Duke University, Durham, NC, 27710, USA

SOURCE: Journal of Biological Chemistry (1998), 273(48), 31880-31889
CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Ca2+/**calmodulin**-dependent protein **kinases** I and IV (CaMKI and CaMKIV, resp.) require phosphorylation on an equivalent single Thr in the activation loop of subdomain VIII for maximal activity. Two distinct CaMKI/IV kinases, CaMKKα and CaMKKβ, were purified from rat **brain** and partially sequenced (Edelman, A. M., Mitchelhill, K., Selbert, M. A., Anderson, K. A., Hook, S. S., Stapleton, D., Goldstein, E. G., Means, A. R., and Kemp, B. E. (1996) J. Biol. Chemical 271, 10806-10810). We report here the **cloning** and sequencing of cDNAs for **human** and rat CaMKKβ, tissue and regional **brain** localization of CaMKKβ protein, and mRNA and functional characterization of **recombinant** CaMKKβ in vitro and in Jurkat T cells. The sequences of **human** and rat CaMKKβ demonstrate 65% identity and 80% similarity with CaMKKα and 30-40% identity with CaMKI and CaMKIV themselves. CaMKKβ is broadly distributed among rat tissues with highest levels in CaMKIV-**expressing** tissues such as **brain**, thymus, spleen, and testis. In **brain**, CaMKKβ tracks more closely with CaMKIV than does CaMKKα. Bacterially **expressed** CaMKKβ undergoes intramol. autophosphorylation, is regulated by Ca2+/CaM, and phosphorylates CaMKI and CaMKIV on Thr177 and Thr200, resp. CaMKKβ activates both CaMKI and CaMKIV when coexpressed in Jurkat T cells as judged by phosphorylated cAMP response element-binding protein-dependent reporter gene **expression**. CaMKKβ activity is enhanced by elevation of intracellular Ca2+, although substantial activity is observed at the resting Ca2+ concentration The strict Ca2+ requirement of CaMKIV-dependent phosphorylation of cAMP response element-binding protein, is therefore controlled at the level of CaMKIV rather than CaMKK.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 6 OF 8 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 94375404 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8089075
TITLE: cDNA **cloning** and **expression** of **human calmodulin**-dependent protein **kinase** IV.
AUTHOR: Kitani T; Okuno S; Fujisawa H
CORPORATE SOURCE: Department of Biochemistry, Asahikawa Medical College, Hokkaido.
SOURCE: Journal of biochemistry, (1994 Apr) 115 (4) 637-40. Journal code: 0376600. ISSN: 0021-924X.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D30742
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941031
Last Updated on STN: 19980206
Entered Medline: 19941018

AB **Calmodulin**-dependent protein **kinase** IV (CaM-kinase IV) is a Ca(2+)-responsive multifunctional protein kinase which occurs abundantly in the **brain** and thymus. A **human** cDNA **clone** encoding CaM-kinase IV was isolated from a Jurkat cell cDNA library and its nucleotide sequence was determined. The cDNA sequence encoded a protein consisting of 473 amino acids with a molecular weight of 51,925. The nucleotide sequence for the coding region and the deduced amino acid sequence showed 81 and 80% identities with those of the rat enzyme, respectively. Western blot analysis, using a polyclonal antibody raised against the **recombinant human** CaM-kinase IV, which was **expressed** in Escherichia coli, revealed two bands corresponding in mobility to molecular weights of 60,000 and 61,000, respectively, in a Jurkat cell extract. The antibody also cross-reacted with both isoforms of CaM-kinase IV from rat cerebellum, the apparent molecular weights being 62,000 and 64,000, respectively.

L10 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:537009 HCAPLUS
DOCUMENT NUMBER: 119:137009
TITLE: **Expression** of a calcium/**calmodulin**-dependent protein **kinase**, CaM kinase-Gr, in **human** T lymphocytes. Regulation of kinase activity by T cell receptor signaling
AUTHOR(S): Hanissian, Silva H.; Frangakis, Maria; Bland, Molly M.; Jawahar, Satya; Chatila, Talal A.
CORPORATE SOURCE: Div. Immunol., Child. Hosp., Boston, MA, 02115, USA
SOURCE: Journal of Biological Chemistry (1993), 268(27), 20055-63
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Ca2+/**calmodulin**-dependent protein **kinase** type Gr (CaM kinase-Gr) is a Ca2+/**calmodulin**-dependent protein **kinase** which is enriched in the **brain** and thymus. In this study, the authors examined the **expression** of CaM kinase-Gr in **human** lymphocytes and the regulation of its catalytic activity by antigen receptor signaling. CaM kinase-Gr was found selectively **expressed** in T lymphocytes in a developmentally regulated manner. It was present at severalfold higher levels in immature thymocytes (CD3low, CD4+CD8+) relative to mature thymocytes (CD3high, CD4+CD8-/CD8+/CD4-) or to circulating T lymphocytes. The kinase was preferentially **expressed** in CD4+ T lymphocytes, but was not detected in B lymphocytes or in monocytes. The impact of T cell antigen receptor-CD3 complex (TCR·CD3) signaling on kinase activity was examined using Jurkat **human** leukemic T lymphocytes as a model. Treatment of

Jurkat cells with anti TCR·CD3 monoclonal antibody induced rapid auto-phosphorylation of the kinase on serine residues and a dramatic, auto-phosphorylation-dependent enhancement of both Ca2+/calmodulin-dependent and autonomous kinase activity. Enzyme auto-phosphorylation and activation were dependent on the influx of extracellular Ca2+ following receptor signaling but could not be induced by an influx of extracellular Ca2+ triggered by ionophores, indicating that addnl. signals delivered via TCR·CD3 contribute to the activation of CaM kinase-Gr. These findings suggest a role for CaM kinase-Gr in T lymphocyte development and activation and indicate the presence of stringent regulatory mechanisms governing the activity of this kinase in situ.

L10 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:403901 HCAPLUS
DOCUMENT NUMBER: 119:3901
TITLE: **Cloning** and analysis of two new isoforms of multifunctional calcium/**calmodulin**-dependent protein **kinase**. **Expression** in multiple **human** tissues
AUTHOR(S): Nghiem, Paul; Saati, Shahin M.; Martens, Christine L.; Gardner, Phyllis; Schulman, Howard
CORPORATE SOURCE: Dep. Pharmacol., Stanford Med. Sch., Stanford, CA, 94305, USA
SOURCE: Journal of Biological Chemistry (1993), 268(8), 5471-9
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Multifunctional Ca2+/**calmodulin**-dependent protein **kinase** (CaM kinase) is a mediator of calcium signals in diverse signaling pathways. In **human** lymphocytes and epithelial tissues, CaM kinase activates a chloride channel via a Ca2+-dependent pathway which is preserved in cystic fibrosis. To characterize the CaM kinase present in these tissues the authors have **cloned** an isoform of this kinase from **human** T lymphocytes. The cDNA sequences of 2 variants of this **human** CaM kinase, γB and γC, are predicted to translate to 518 and 495 amino acids, resp. Amino acid differences between these isoforms and the rat **brain** γ isoform (which the authors refer to as γA) are localized to the variable domain. RNase protection of this variable region revealed the level of **expression** of γB and γC CaM kinase mRNAs in nine **human** tissues and cell lines. When transfected into Jurkat T cells, the γB cDNA encoded a functional kinase which cosedimented on sucrose gradients with endogenous T cell CaM kinase activity and formed a large multimeric enzyme. The **recombinant** γB isoform displayed 2 phases of autophosphorylation characteristic of CaM kinases, including the phase which converts it to a partially Ca2+-independent species. Site-directed mutagenesis of the predicted autoinhibitory domain yielded a mutant which was .apprx.37% active in the absence of Ca2+/calmodulin, confirming the region as critical for autoregulation, and suggesting this mutant as a tool for studying the role of CaM kinase in nonneuronal tissues.

=> e yan c/au
E1 1 YAN BUYU/AU
E2 1 YAN BY ZHANQING/AU
E3 996 --> YAN C/AU
E4 2 YAN C B/AU
E5 123 YAN C C/AU
E6 5 YAN C C S/AU
E7 3 YAN C CHAN/AU
E8 14 YAN C D/AU
E9 1 YAN C D L/AU
E10 17 YAN C F/AU

```
E11          44      YAN C G/AU
E12         439      YAN C H/AU

=> s e3
L11         996 "YAN C"/AU

=> e ketchum k/au
E1           12      KETCHUM JR L H/AU
E2            1      KETCHUM JR R L/AU
E3           34 --> KETCHUM K/AU
E4          220      KETCHUM K A/AU
E5            1      KETCHUM K J/AU
E6           32      KETCHUM K L/AU
E7           20      KETCHUM KAREN/AU
E8          172      KETCHUM KAREN A/AU
E9            1      KETCHUM KAREN ANN/AU
E10           2      KETCHUM KATHY/AU
E11           4      KETCHUM KEVIN/AU
E12           3      KETCHUM KEVIN L/AU

=> s e7-e9
L12         193 ("KETCHUM KAREN"/AU OR "KETCHUM KAREN A"/AU OR "KETCHUM KAREN
                ANN"/AU)

=> e merkulov g/au
E1            1      MERKULOV EVGENIY/AU
E2            4      MERKULOV F N/AU
E3           25 --> MERKULOV G/AU
E4           10      MERKULOV G A/AU
E5            2      MERKULOV G M/AU
E6           75      MERKULOV G V/AU
E7           24      MERKULOV GENNADY/AU
E8           54      MERKULOV GENNADY V/AU
E9            1      MERKULOV GENNAY V/AU
E10           1      MERKULOV GP/AU
E11           1      MERKULOV GRIGORY VALERIEVITCH/AU
E12           3      MERKULOV I/AU

=> s e7-e9
L13          79 ("MERKULOV GENNADY"/AU OR "MERKULOV GENNADY V"/AU OR "MERKULOV
                GENNAY V"/AU)

=> e beasley e m/au
E1            1      BEASLEY E H/AU
E2            6      BEASLEY E L/AU
E3          291 --> BEASLEY E M/AU
E4            7      BEASLEY E O/AU
E5            1      BEASLEY E S G/AU
E6            2      BEASLEY E T/AU
E7            4      BEASLEY E W/AU
E8            2      BEASLEY E W 3RD/AU
E9            2      BEASLEY E W III/AU
E10           1      BEASLEY E W JR/AU
E11           1      BEASLEY EDWARD E/AU
E12           1      BEASLEY EDWARD EVANS/AU

=> s e3
L14         291 "BEASLEY E M"/AU

=> d his

     (FILE 'HOME' ENTERED AT 08:12:09 ON 04 MAY 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
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     LIFESCI' ENTERED AT 08:12:39 ON 04 MAY 2004
L1       2087394 S CALCIUM OR CALMODULIN
L2         38966 S L1 (2W)KINASE?
L3          8783 S HUMAN AND L2
L4       6504693 S CLON? OR EXPRESS? OR RECOMBINANT
L5          4552 S L3 AND L4
L6       4062032 S BRAIN OR LUNG OR HIPPCAMPUS
L7           771 S L5 AND L6
L8            78 S HUMAN(W)L2
L9            17 S L7 AND L8
L10            8 DUP REM L9 (9 DUPLICATES REMOVED)
                 E YAN C/AU
L11          996 S E3
                 E KETCHUM K/AU
L12          193 S E7-E9
                 E MERKULOV G/AU
L13           79 S E7-E9
                 E BEASLEY E M/AU
L14          291 S E3

=> s l11 or l12 or l13 or l14
L15       1449 L11 OR L12 OR L13 OR L14

=> s l3 and l15
L16           5 L3 AND L15

=> dup rem l16
PROCESSING COMPLETED FOR L16
L17           4 DUP REM L16 (1 DUPLICATE REMOVED)

=> d 1-4 ibib ab

L17   ANSWER 1 OF 4  BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
ACCESSION NUMBER: 2002-17807  BIOTECHDS
TITLE:            Nucleic acid molecules encoding calcium/calmodulin
                  -dependent protein kinases, useful for preventing
                  diagnosing and treating e.g. cancers, psoriasis and
                  inflammation;
                     recombinant protein production by vector-mediated gene
                     transfer and expression in host cell, useful for gene
                     therapy
AUTHOR:           YE J; YAN C; DI FRANCESCO V; BEASLEY E M
PATENT ASSIGNEE:  PE CORP NY
PATENT INFO:      US 6387677 14 May 2002
APPLICATION INFO: US 2001-800960 8 Mar 2001
PRIORITY INFO:    US 2001-800960 8 Mar 2001
DOCUMENT TYPE:    Patent
LANGUAGE:         English
OTHER SOURCE:     WPI: 2002-478444 [51]
AB    DERWENT ABSTRACT:
      NOVELTY - An isolated nucleic acid molecule (I) encoding a calcium/
      calmodulin-dependent protein kinase, is new.
           DETAILED DESCRIPTION - An isolated nucleic acid molecule (I)
      encoding a calcium/calmodulin-dependent protein kinase
      , comprising a nucleotide sequence selected from: (a) a nucleotide
      sequence that encodes a protein comprising a fully defined 565 amino acid
      sequence (A1) given in the specification; (b) a nucleotide sequence
      comprising the fully defined 2061 nucleotide sequence (N1) given in the
      specification ((N1) is a complementary deoxyribonucleic acid (cDNA)
      encoding the kinase); and/or (c) a nucleotide sequence comprising the
      defined 62804 nucleotide sequence (N2) given in the specification ((N2)
      is a genomic sequence that spans the gene encoding the kinase protein).
      INDEPENDENT CLAIMS are also included for: (1) a nucleic acid vector (II)
      comprising (I); (2) a host cell (III) containing the vector (II); (3)
```

producing (IV) a polypeptide comprising (A1), comprising culturing the host cell (III) under conditions sufficient for the production of said polypeptide, and recovering said polypeptide from the host cell culture; and (4) an isolated nucleic acid molecule (I') comprising a nucleotide sequence that is completely complementary to (I).

BIOTECHNOLOGY - Preferred Vectors: The vector (II) is a plasmid, virus or bacteriophage. (I) is inserted into the vector in proper orientation and correct reading frame so that the protein of (A1) may be expressed by a cell transformed with the vector. The isolated nucleic acid molecule may be operatively linked to a promoter sequence. Preparation: (I) and the protein it encodes may be produced via standard recombinant and synthetic methodologies e.g. by culturing (IV) the cell (III) (claimed).

ACTIVITY - Cytostatic; Anti-inflammatory; Anti-arteriosclerotic; Anti-psoriatic. No biological data given.

MECHANISM OF ACTION - Gene therapy; Protein therapy; Vaccine; Enzymatic (calcium/**calmodulin**-dependent protein **kinase**). The gene (I) and encoded protein are related to the family of calcium/**calmodulin**-dependent protein **kinases**, which are serine/threonine kinases. The protein shows a particularly high degree of similarity to calcium/**calmodulin**-dependent protein **kinase** II (CaM II). CaM II is comprised of alpha, beta, gamma, and delta subunits. Each subunit is encoded by a separate gene and alternatively splice forms of each subunit have been found (Breen et al., Biochem. Biophys. Res. Commun. 236 (2), 473-478 (1997)). CaM II exerts important effects on hormones and neurotransmitters that utilize calcium as a second messenger. Beta-cell CaM II activity is associated with insulin secretion, and multiple isoforms of CaM II are expressed in **human** islets of Langerhans (Breen et al., Biochem. Biophys. Res. Commun. 236 (2), 473-478 (1997)). It has been suggested that CaM II controls activation-induced cellular differentiation, and is important for imparting antigen-dependent memory to T cells (Bui et al., Cell 100: 457-467, 2000).

USE - These polynucleotide sequences (I) and the peptides they encode can be used as models for the development of **human** therapeutic targets, aid in the identification of therapeutic proteins, and serve as targets for the development of **human** therapeutic agents that modulate kinase activity in cells and tissues that express the kinase. The calcium/**calmodulin**-dependent protein **kinase** encoded by (I) is expressed in **humans** in the placenta, breast cancers (including mammary adenocarcinoma), skin melanotic melanomas, ovary adenocarcinomas, uterus leiomyosarcomas, Burkitt's lymphomas (lymph), duodenal adenocarcinomas (small intestine), and fetal brain tumors and in disease conditions including inflammation, arteriosclerosis, and psoriasis (claimed).

ADMINISTRATION - Standard methodologies.

ADVANTAGE - Kinase proteins, particularly members of the calcium/**calmodulin**-dependent protein **kinase** subfamily, are a major target for drug action and development. Accordingly, it is valuable to the field of pharmaceutical development to identify and characterize previously unknown members of this subfamily of kinase proteins. (I) Encodes a previously unidentified **human** kinase protein that has homology to members of the calcium/**calmodulin**-dependent protein **kinase** subfamily.

EXAMPLE - No suitable example given.(85 pages)

L17 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:778135 HCAPLUS
DOCUMENT NUMBER: 137:290019
TITLE: Protein, gene and cDNA sequences of a novel **human** protein kinase related to calcium-**calmodulin**-dependent protein **kinase** and their uses in drug screening
INVENTOR(S): Shao, Wei; **Merkulov, Gennady V.**; Di

Francesco, Valentina
PATENT ASSIGNEE(S): PE Corporation (NY), USA; Beasley, Ellen M.
SOURCE: PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002079431	A2	20021010	WO 2002-US9744	20020401
WO 2002079431	A3	20030313		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003140354	A1	20030724	US 2001-820790	20010330
US 6716604	B2	20040406		
EP 1383873	A2	20040128	EP 2002-757858	20020401
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: US 2001-820790 A 20010330
WO 2002-US9744 W 20020401

AB The invention provides protein, cDNA and genomic sequences for a novel **human** protein kinase related to calcium-**calmodulin** -dependent protein **kinase**. Specifically, a virtual northern blot shows calcium-**calmodulin**-dependent protein **kinase** gene expression in the fetal brain, testis, lung small cell carcinoma, and uterus endometrium adenocarcinoma. Twenty six single nucleotide polymorphism has been found on calcium-**calmodulin**-dependent protein **kinase** gene that has been mapped to chromosome 5. The invention also relates to screening modulator of calcium-**calmodulin**-dependent protein **kinase** and use them in therapy. The invention further relates to methods, vector and hosts for expression of calcium-**calmodulin**-dependent protein **kinase**.

L17 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:755098 HCAPLUS
DOCUMENT NUMBER: 137:274169
TITLE: Protein, gene and cDNA sequences of a novel **human** protein kinase related to synthase and their uses in drug screening
INVENTOR(S): **Merkulov, Gennady V.**; Gong, Fangcheng; Di Francesco, Valentina; Beasley, Ellen M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 40 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002142427	A1	20021003	US 2001-817181	20010327
WO 2002077192	A2	20021003	WO 2002-US9326	20020327

WO 2002077192 A3 20040129
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
US 2003077799 A1 20030424 US 2002-300828 20021121
PRIORITY APPLN. INFO.: US 2001-817181 A 20010327

AB The invention provides protein, cDNA and genomic sequences for a novel **human** protein kinase related to calcium/**calmodulin** -dependent protein **kinase** I. The calcium/**calmodulin** -dependent protein **kinase** I gene is expressed in **human** placenta, kidney, cervix, eye, colon, liver and whole brain. Ten single nucleotide polymorphism has been found on calcium/**calmodulin** -dependent protein **kinase** I gene mapped to chromosome 3. The invention also relates to screening modulator of calcium/ **calmodulin**-dependent protein **kinase** I and use them in therapy. The invention further relates to methods, vector and hosts for expression of calcium/**calmodulin**-dependent protein **kinase** I.

L17 ANSWER 4 OF 4 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
ACCESSION NUMBER: 2002-07499 BIOTECHDS
TITLE: New **calmodulin**-binding **kinase** peptides and nucleic acid encoding the peptides, useful as models for developing **human** therapeutic targets or in screening for compounds that modulate kinase; **human** recombinant enzyme production useful for drug target, drug screening, and ribozyme and antisense gene therapy
AUTHOR: **YAN C**; WEI M; KETCHUM K; MERKULOV G; **BEASLEY E M**
PATENT ASSIGNEE: APPLERA CORP
PATENT INFO: WO 2001092492 6 Dec 2001
APPLICATION INFO: WO 2000-US17327 30 May 2000
PRIORITY INFO: US 2000-734030 12 Dec 2000
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2002-097770 [13]

AB DERWENT ABSTRACT:
NOVELTY - A new isolated peptide (I) having a sequence comprising: (a) a 501-amino acid sequence (P1) defined in the specification; (b) an allelic variant or ortholog of P1 encoded by a nucleic acid that hybridizes under stringent conditions to the opposite strand of a 3124 (S1) or 7542 (S2) base pair sequence defined in the specification; or (c) a fragment of P1 comprising at least 10 contiguous amino acids.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) an isolated antibody that selectively binds to (I); (2) an isolated nucleic acid (II) having a sequence which: (a) encodes P1; (b) encodes an allelic variant or ortholog of P1, where the nucleotide sequence hybridizes under stringent conditions to the opposite strand of S1 or S2; (c) encodes a fragment of P1 comprising at least 10 contiguous amino acids; or (d) is a complement of (a)-(c); (3) a gene chip comprising (II); (4) a transgenic non-**human** animal comprising (II); (5) a nucleic acid vector comprising (II); (6) a host cell containing the vector; (7) a method for producing (I) by introducing a nucleotide sequence encoding an amino acid sequences defined above into a host cell, and culturing the host cell under conditions for the expression of the peptides from the nucleotide sequence; (8) a method of detecting the presence of (I) in a sample by contacting the sample with a

detection agent that specifically detects the presence of (I) in the sample; (9) a method for detecting the presence of (II) in a sample by contacting the sample with an oligonucleotide that hybridizes to (II) under stringent conditions, and determining if the oligonucleotide binds to (II); (10) methods for identifying a modulator of (I) by contacting the peptide or a cell expressing (I) with an agent and determining if the agent has modulated the function or activity of the peptide; (11) a method for identifying an agent that binds to (I) by contacting (I) with an agent and assaying the contacted mixture to determine whether a complex is formed with the agent bound to the peptide; (12) a pharmaceutical composition comprising an agent and a carrier; (13) a method for treating a disease or condition mediated by a **human** kinase protein by administering to a patient an agent identified by the method of (11); (14) an isolated **human** kinase peptide having a sequence that shares at least 70% homology with P1; and (15) an isolated nucleic acid molecule encoding a **human** kinase peptide sharing at least 80% homology with S1 or S2.

BIOTECHNOLOGY - Preferred Method: In identifying a modulator of (I), the agent is administered to a host cell comprising an expression vector that expresses (I). Preferred Sequence: The isolated **human** kinase peptide shares at least 90% homology with P1. The nucleic acid encoding the **human** kinase peptide preferably shares at least 90% homology with S1 or S2.

ACTIVITY - None given in the source document.

MECHANISM OF ACTION - Calmodulin modulator.

USE - The peptide and nucleic acid sequences can be used as models for the development of **human** therapeutic targets, in the identification of therapeutic proteins, and serve as targets for the development of **human** therapeutic agents that modulate kinase activity in cells and tissues that express the kinase. The peptides or proteins are useful for raising antibodies, as a reagent in assays to determine the levels of the protein in biological fluids, as markers for tissues in which the corresponding protein is expressed, and in screening for compounds that inhibit or stimulate interaction between kinase protein and a molecule that normally interacts with the kinase protein. The nucleic acids can be used as probes or primers, for constructing recombinant vectors, for designing ribozymes corresponding to all or a part of the mRNA produced from the nucleic acid, for constructing host cells or transgenic animals, for treating a disorder associated with nucleic acid expression of the kinase gene, particularly biological and pathological processes that are mediated by the kinase in cells and tissues that express it, as antisense constructs to control kinase gene expression in cells, tissues or organisms, and in gene therapy.

EXAMPLE - No examples given. (75 pages)

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NOTICE SET TO 1 U.S. DOLLAR FOR DISPLAY COMMAND
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=> index IFICLS,PATOSEP,PATDPA,INPADOC

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	52.36	52.57

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CA SUBSCRIBER PRICE	-5.54	-5.54

INDEX 'IFICLS, PATOSEP, PATDPA, INPADOC' ENTERED AT 08:25:38 ON 04 MAY 2004

4 FILES IN THE FILE LIST IN STNINDEX

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search error messages that display as 0* with SET DETAIL OFF.

=>  S US 6387677/PN,APPS

          1    FILE IFICLS
          1    FILE INPADOC

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L18 QUE US 6387677/PN,APPS

=> FILE HITS

COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.57      53.14

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)            SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                       0.00      -5.54

FILE 'IFICLS' ENTERED AT 08:25:41 ON 04 MAY 2004
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=>  S L18

L19          2 L18

=>  D IBIB LS 1-

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DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:y

L19  ANSWER 1 OF 2  IFICLS  COPYRIGHT 2004 IFI on STN

ACCESSION NO.:         3682610  IFICLS
PATENT ASSIGNEE:       Applera Corp
PATENT INFORMATION:    US  6387677  20020514
DOCUMENT TYPE:         REASSIGNED
REASSIGNMENT INFO.:
  DATE:                20030325
  KIND:                ASSIGNMENT OF ASSIGNORS INTEREST
  ASSIGNOR:            YE, JANE, DATE SIGNED: 05/30/2002 ; YAN, CHUNHUA, DATE
                       SIGNED: 01/31/2003 ; DIFRANCESCO, VALENTINA, DATE SIGNED:
                       03/24/2003 ; BEASLEY, ELLEN M., DATE SIGNED: 03/24/2003
  ASSIGNEE:            APPLERA CORPORATION, 301 MERRITT 7, P.O. BOX 5435,
                       NORWALK, CONNECTICUT, 06856-5435
  AGENT:               CELERA GENOMICS CORP., ATTN: WAYNE MONTGOMERY, VICE PRES,
                       INTEL, 45 WEST GUDE DRIVE, C2-4#20, ROCKVILLE, MD 20850
  MICROFILM REEL NO:   013882
  MICROFILM FRAME NO:  0634

L19   ANSWER 2 OF 2  INPADOC  COPYRIGHT 2004 EPO on STN

LEVEL 1
ACCESSION NUMBER:        176005975 INPADOC   EW 200223  ED 20020611
                                             UW 200306  UP 20030210
TITLE:                   NUCLEIC ACID MOLECULES ENCODING HUMAN
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      CALCIUM/CALMODULIN (CAMK) DEPENDENT KINASE PROTEINS.
INVENTOR(S):
  ORIGINAL:               YE JANE; YAN CHUNHUA; DI FRANCESCO VALENTINA; BEASLEY
                          ELLEN M.
  STANDARDIZED:           YE JANE; YAN CHUNHUA; DI FRANCESCO VALENTINA; BEASLEY
                          ELLEN M
  LOCATION:               US; US; US; US
PATENT ASSIGNEE(S):
  ORIGINAL:               PE CORPORATION (NY)
  STANDARDIZED:           PE CORP NY
  LOCATION:               US
DOCUMENT TYPE:            Patent
PATENT INFO. TYPE:        USBA PATENT (NO PREVIOUS PRE-GRANT PUBLICATION)
PATENT INFORMATION:
                          NUMBER              KIND     DATE
                          ---------------------------------
                            US 6387677           BA 20020514
APPLICATION INFO.:        US 2001-800960       A  20010308
PRIORITY APPLN. INFO.:    US 2001-800960       A  20010308


=> SET NOTICE LOGIN DISPLAY

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=> d his

     (FILE 'HOME' ENTERED AT 08:12:09 ON 04 MAY 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 08:12:39 ON 04 MAY 2004
L1       2087394 S CALCIUM OR CALMODULIN
L2         38966 S L1 (2W)KINASE?
L3          8783 S HUMAN AND L2
L4       6504693 S CLON? OR EXPRESS? OR RECOMBINANT
L5          4552 S L3 AND L4
L6       4062032 S BRAIN OR LUNG OR HIPPCAMPUS
L7           771 S L5 AND L6
L8            78 S HUMAN(W)L2
L9            17 S L7 AND L8
L10            8 DUP REM L9 (9 DUPLICATES REMOVED)
                 E YAN C/AU
L11          996 S E3
                 E KETCHUM K/AU
L12          193 S E7-E9
                 E MERKULOV G/AU
L13           79 S E7-E9
                 E BEASLEY E M/AU
L14          291 S E3
L15         1449 S L11 OR L12 OR L13 OR L14
L16            5 S L3 AND L15
L17            4 DUP REM L16 (1 DUPLICATE REMOVED)
                 SET NOTICE DISPLAY 1

     INDEX 'IFICLS, PATOSEP, PATDPA, INPADOC' ENTERED AT 08:25:38 ON 04 MAY
     2004
                 SEA US 6387677/PN,APPS
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                1   FILE IFICLS
                1   FILE INPADOC
L18              QUE US 6387677/PN,APPS
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     FILE 'IFICLS, INPADOC' ENTERED AT 08:25:41 ON 04 MAY 2004
L19            2 S L18
                 SET NOTICE LOGIN DISPLAY

=> dup rem l8
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    12.09      65.23

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00      -5.54

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FILE 'BIOSIS' ENTERED AT 08:28:57 ON 04 MAY 2004
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FILE 'LIFESCI' ENTERED AT 08:28:57 ON 04 MAY 2004
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PROCESSING COMPLETED FOR L8
L20           68 DUP REM L8 (10 DUPLICATES REMOVED)

=> d 1-68 ibib ab

L20  ANSWER 1 OF 68     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   2004091023     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 14980499
TITLE:              Effects of PU.1-induced mouse calcium-calmodulin-dependent
                    kinase I-like kinase (CKLiK) on apoptosis of murine
                    erythroleukemia cells.
AUTHOR:             Yamada Toshiyuki; Suzuki Mitsuhiro; Satoh Hitoshi;
                    Kihara-Negishi Fumiko; Nakano Hiroyasu; Oikawa Tsuneyuki
CORPORATE SOURCE:   Department of Cell Genetics, Sasaki Institute, Tokyo
                    101-0062, Japan.. oikawa@sasaki.or.jp
SOURCE:             Experimental cell research, (2004 Mar 10) 294 (1) 39-50.
                    Journal code: 0373226. ISSN: 0014-4827.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200403
ENTRY DATE:         Entered STN: 20040225
                    Last Updated on STN: 20040331
                    Entered Medline: 20040330
AB   PU.1, a hematopoietic cell-specific Ets family transcription factor, is
     involved in the generation of murine erythroleukemia (MEL). To identify
     the target gene(s) of PU.1 in MEL cells, we carried out differential
     display (DD) analysis and isolated a novel gene whose expression was
     up-regulated after overexpression of PU.1 in MEL cells. Because the gene
     exhibited about 90% homology with the human calcium
     -calmodulin-dependent kinase I-like kinase (CKLiK) gene, it was
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identified as a mouse homologue of human CKLiK. The mCKLiK gene was mapped to the mouse chromosome 2A1-A3 region and shown to be expressed predominantly in T cells lymphoma and embryonal carcinoma cell lines and primary thymus and brain. Two types of transcripts were present showing a difference in the 3' portion of the coding region and CREB-activating ability. Overexpression of each isoform of mCKLiK in MEL cells revealed that one of them induces, while the other inhibits apoptosis under low serum condition. Differentiation inhibition and lineage switch to myelomonocytes, which were previously observed in MEL cells overexpressing PU.1, were not provoked in the cells overexpressing mCKLiK. These results suggest that mCKLiK is up-regulated by PU.1 in MEL cells and involved in apoptosis of the cells.

L20 ANSWER 2 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2004:68086 BIOSIS
DOCUMENT NUMBER: PREV200400068613
TITLE: Isolated human calcium/calmodulin (CaMk) dependent kinase proteins.
AUTHOR(S): Ye, Jane [Inventor, Reprint Author]; Yan, Chunhua [Inventor]; Di Francesco, Valentina [Inventor]; Beasley, Ellen M. [Inventor]
CORPORATE SOURCE: ASSIGNEE: Applera Corporation
PATENT INFORMATION: US 6664085 December 16, 2003
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Dec 16 2003) Vol. 1277, No. 3. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133 (ISSN print).
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Jan 2004
Last Updated on STN: 28 Jan 2004
AB The present invention provides amino acid sequences of peptides that are encoded by genes within the human genome, the kinase peptides of the present invention. The present invention specifically provides isolated peptide and nucleic acid molecules, methods of identifying orthologs and paralogs of the kinase peptides, and methods of identifying modulators of the kinase peptides.

L20 ANSWER 3 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:1006038 HCAPLUS
DOCUMENT NUMBER: 140:144472
TITLE: HIV-1 Tat induces TNF-α production by human monocyte: Involvement of calcium PKC pathways
AUTHOR(S): Contreras, Xavier; Bennasser, Yamina; Chazal, Nathalie; Bahraoui, Elmostafa
CORPORATE SOURCE: Laboratoire d'immuno-virologie, EA3038, Universite Paul Sabatier 118, Toulouse, 31062, Fr.
SOURCE: Journal de la Societe de Biologie (2003), 197(3), 267-275
CODEN: JDSBFG; ISSN: 1295-0661
PUBLISHER: Masson Editeur
DOCUMENT TYPE: Journal
LANGUAGE: French
AB Here, the authors investigated the signaling pathways triggered by Tat in human monocyte to induce TNF-α. In monocytes, calcium, PKA, and PKC pathways are highly implicated in the expression of cytokine genes. The authors' data show that (1) extracellular calcium is required for the calcium signal initiated by Tat in the monocyte and is required for TNF-α production, PKC pathway is also required, whereas the PKA pathway does not seem to be involved (2) downstream from PKC, activation of NFκB is essential while ERK1/2 MAP kinases, even though activated by Tat, are not directly involved in the pathway signaling leading to TNF-α production
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 4 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:156649 HCAPLUS
DOCUMENT NUMBER: 139:50301
TITLE: Ca2+/calmodulin-dependent translocation of sphingosine kinase: Role in plasma membrane relocation but not activation
AUTHOR(S): Young, Kenneth W.; Willets, Jonathon M.; Parkinson, M. Janine; Bartlett, Paula; Spiegel, Sarah; Nahorski, Stefan R.; Challiss, R. A. John
CORPORATE SOURCE: Department of Cell Physiology and Pharmacology, University of Leicester, Leicester, LE1 9HN, UK
SOURCE: Cell Calcium (2003), 33(2), 119-128
CODEN: CECADV; ISSN: 0143-4160
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Activation of sphingosine kinase (SPHK), thereby increasing cellular levels of sphingosine 1-phosphate (S1P), may be involved in a variety of intracellular responses including Ca2+ signaling. This study uses mammalian SPHK1a, tagged with enhanced green fluorescent protein (eGFP), to examine whether translocation of this enzyme is linked with Ca2+-mobilizing responses. Real-time confocal imaging of SPHK1a-eGFP in human SH-SY5Y neuroblastoma cells visualized a relocation of the enzyme from the cytosol to the plasma membrane in response to Ca2--mobilizing stimuli (muscarinic M3- or lysophosphatidic acid receptor activation, and thapsigargin-mediated store release). This redistribution was preceded by a transient increase in cytosolic SPHK1a-eGFP levels due to liberation of SPHK from localized higher intensity regions. Translocation was dependent on Ca2+ mobilization from intracellular stores, and was prevented by pretreatment with the Ca2+/calmodulin inhibitor W-7, but not W-5 or KN-62. In functional studies, pretreatment with W-7 lowered basal and M3-receptor-mediated cellular S1P production However, this pretreatment did not alter agonist-mediated Ca2+ responses, and SPHK1a-eGFP activity itself appeared insensitive to Ca2+/calmodulin and W-7. These data suggest a role for Ca2+/calmodulin in controlling the subcellular distribution but not the activity of SPHK1a.
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 5 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:448303 BIOSIS
DOCUMENT NUMBER: PREV200300448303
TITLE: Identification and characterization of novel components of a Ca2+/calmodulin-dependent protein kinase cascade in HeLa cells.
AUTHOR(S): Ishikawa, Yumi; Tokumitsu, Hiroshi [Reprint Author]; Inuzuka, Hiroyuki; Murata-Hori, Maki; Hosoya, Hiroshi; Kobayashi, Ryoji
CORPORATE SOURCE: Department of Signal Transduction Sciences, Kagawa Medical University, 1750-1 Miki-cho, Kita-gun, Takamatsu, Kagawa, 761-0793, Japan
tokumit@kms.ac.jp
SOURCE: FEBS Letters, (28 August 2003) Vol. 550, No. 1-3, pp. 57-63. print.
CODEN: FEBLAL. ISSN: 0014-5793.
DOCUMENT TYPE: Article
LANGUAGE: English
OTHER SOURCE: DDBJ-AB081336; EMBL-AB081336; GenBank-AB081336; DDBJ-AB081337; EMBL-AB081337; GenBank-AB081337; DDBJ-AB081726; EMBL-AB081726; GenBank-AB081726
ENTRY DATE: Entered STN: 1 Oct 2003
Last Updated on STN: 1 Oct 2003

AB In this report, we cloned a novel calmodulin-kinase (CaM-KIdelta) from HeLa cells and characterized its activation mechanism. CaM-KIdelta exhibits Ca2+/CaM-dependent activity that is enhanced (apprx30-fold) in vitro by phosphorylation of its Thr180 by CaM-K kinase (CaM-KK)alpha, consistent with detection of CaM-KIdelta-activating activity in HeLa cells. We also identified a novel CaM-KKbeta isoform (CaM-KKbeta-3) in HeLa cells whose activity was highly Ca2+/CaM-independent. Transiently expressed CaM-KIdelta exhibited enhanced protein kinase activity in HeLa cells without ionomycin stimulation. This sustained activation of CaM-KIdelta was completely abolished by Thr180Ala mutation and inhibited by CaM-KK inhibitor, STO-609, indicating a functional CaM-KK/CaM-KIdelta cascade in HeLa cells.

L20 ANSWER 6 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2004:54040 BIOSIS
DOCUMENT NUMBER: PREV200400051442
TITLE: Microarray gene expression profiling in patients with congestive cardiomyopathy and pressure overload hypertrophy due to aortic stenosis.
AUTHOR(S): Bartunek, Jozef [Reprint Author]; Kong, Sek W.; Vanderheyden, Marc [Reprint Author]; Brown, Jeffrey; Riggy, Lauren; Tack, Wouter [Reprint Author]; Goethals, Marc [Reprint Author]; Casselman, Filip; Wellens, Francis; de Bruyne, Bernard [Reprint Author]; Izumo, Seigo; Schinke, Martina
CORPORATE SOURCE: Cardiovascular Cntr, Aalst, Belgium
SOURCE: Circulation, (October 28 2003) Vol. 108, No. 17 Supplement, pp. IV-369. print.
Meeting Info.: American Heart Association Scientific Sessions 2003. Orlando, FL, USA. November 09-12, 2003. American Heart Association.
ISSN: 0009-7322 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Jan 2004
Last Updated on STN: 21 Jan 2004

L20 ANSWER 7 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
ACCESSION NUMBER: 2002:337571 BIOSIS
DOCUMENT NUMBER: PREV200200337571
TITLE: Nucleic acid molecules encoding human calcium/calmodulin (CaMK) dependent kinase proteins.
AUTHOR(S): Ye, Jane [Inventor]; Yan, Chunhua [Inventor]; Di Francesco, Valentina [Inventor]; Beasley, Ellen M. [Inventor]
CORPORATE SOURCE: ASSIGNEE: PE Corporation (NY)
PATENT INFORMATION: US 6387677 May 14, 2002
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (May 14, 2002) Vol. 1258, No. 2. http://www.uspto.gov/web/menu/patdata.html. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jun 2002
Last Updated on STN: 12 Jun 2002
AB The present invention provides amino acid sequences of peptides that are encoded by genes within the human genome, the kinase peptides of the present invention. The present invention specifically provides isolated peptide and nucleic acid molecules, methods of identifing orthologs and paralogs of the kinase peptides, and methods of identifying modulators of the kinase peptides.

L20 ANSWER 8 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:658936 HCAPLUS
DOCUMENT NUMBER: 138:236807
TITLE: Participation of the calcium/calmodulin-dependent kinases in hydrogen peroxide-induced IκB phosphorylation in human T lymphocytes
AUTHOR(S): Howe, Christopher J.; LaHair, Michelle M.; Maxwell, Jill A.; Lee, John T.; Robinson, Penni J.; Rodriguez-Mora, Oswaldo; McCubrey, James A.; Franklin, Richard A.
CORPORATE SOURCE: Department of Microbiology and Immunology, Brody School of Medicine, East Carolina University, Greenville, NC, 27858, USA
SOURCE: Journal of Biological Chemistry (2002), 277(34), 30469-30476
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB NF-κB is an important transcription factor that has a role in a variety of responses such as inflammation, oncogenesis, apoptosis, and viral replication. Oxidative stress is well known to induce the activation of NF-κB. Cells can be exposed to either endogenously produced oxidants or oxidants produced by surrounding cells. In addition, ischemia reperfusion and certain cancer therapies such as chemotherapy and photodynamic therapy are thought to result in oxygen radical production Because of the important role that NF-κB has in multiple responses, it is critical to determine the mechanisms by which oxidative stress induces NF-κB activity. We report that the calmodulin antagonist W-7 and the calcium/calmodulin-dependent (CaM) kinase inhibitors KN-93 and K252a, can block oxidative stress-induced IκB phosphorylation in Jurkat T lymphocytes. Furthermore, KN-93 but not KN-92 can block hydrogen peroxide-induced Akt and IKK phosphorylation. In addition, we found that expression of a kinase-dead CaM-KIV construct in two cell lines inhibits IκB phosphorylation or degradation and that expression of CaM-KIV augments hydrogen peroxide-induced IκB phosphorylation and degradation Although the CaM kinases appear to be required for this response, increases in intracellular calcium do not appear to be required. These results identify the CaM kinases as potential targets that can be used to minimize NF-κB activation in response to oxidative stress.

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 9 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:886475 HCAPLUS
DOCUMENT NUMBER: 136:32798
TITLE: Protein, gene and cDNA sequences of **human calmodulin**-binding protein **kinase** sequence homolog
INVENTOR(S): Yan, Chunhua; Wei, Ming-hui; Ketchum, Karen; Merkulov, Gennady; Beasley, Ellen M.
PATENT ASSIGNEE(S): Applera Corporation, USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001092492	A2	20011206	WO 2001-US17327	20010530
WO 2001092492	A3	20020704		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2002015987	A1	20020207	US 2000-734030	20001212
US 6461846	B2	20021008		
EP 1290188	A2	20030312	EP 2001-948244	20010530
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003534794	T2	20031125	JP 2002-500684	20010530
US 2002142430	A1	20021003	US 2002-153921	20020524
US 6653116	B2	20031125		
US 2004038363	A1	20040226	US 2003-669689	20030925

PRIORITY APPLN. INFO.:

US 2000-207281P	P	20000530
US 2000-734030	A	20001212
WO 2001-US17327	W	20010530
US 2002-153921	A3	20020524

AB The invention provides protein and cDNA and genomic sequences for a novel human protein kinase, which has strong sequence homol. with rat, fish and Drosophila calmodulin-binding protein kinases. The gene is expressed in the fetal and adult brain, lung, and hippocampus. Nine single nucleotide polymorphism sites (beyond the ORF or in intron regions) were identified. Thus, the present invention specifically provides isolated peptide and nucleic acid mols., methods of identifying orthologs and paralogs of the protein kinase peptides, methods of identifying modulators of the protein kinase peptides, and methods of diagnosis and treatment of diseases associated with the protein kinase.

L20 ANSWER 10 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:	2001:815942 HCAPLUS
DOCUMENT NUMBER:	136:83769
TITLE:	Effects of calcium, calmodulin, protein kinase C and protein tyrosine kinases on volume-activated taurine efflux in human erythroleukemia cells
AUTHOR(S):	Huang, Chiun-Chien; Chang, Chirn-Bin; Liu, Jer-Yuh; Basavappa, Srisaila; Lim, Poh-Hong
CORPORATE SOURCE:	Department of Physiology, Chung Shan Medical and Dental College, Taichung, 40203, Taiwan
SOURCE:	Journal of Cellular Physiology (2001), 189(3), 316-322 CODEN: JCLLAX; ISSN: 0021-9541
PUBLISHER:	Wiley-Liss, Inc.
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB The effects of calcium, calmodulin, protein kinase C (PKC) and protein tyrosine kinase (PTK) modulators were examined on the volume-activated taurine efflux in the erythroleukemia cell line K562. Exposure to hypoosmotic solution significantly increased taurine efflux and intracellular calcium concentration ([Ca2+]i). The Ca2+ channel blockers La3+ (1 mM), verapamil (200 μM) and nifedipine (100 μM) inhibited the hypoosmotically-induced [Ca2+]i increase by more than 90%, while the volume-activated taurine efflux was inhibited by 61.3±9.5, 74.1±9.3 and 38.0±1.5%, resp. Furthermore, the calmodulin inhibitors W7 (50 μM) and trifluoperazine (10 μM) and the Ca2+/calmodulindependent protein kinase II inhibitor KN-62 (2 μM) significantly blocked the volume-activated taurine efflux by 93.4±2.7, 77.9±3.5 and 61.3±15.8%, resp. In contrast, the PKC inhibitor staurosporine (200 nM) or the PKC activator phorbol 12-myristate 13-acetate (100 nM) did not have significant effects on the volume-activated taurine efflux. However, pretreatment with PTK inhibitors genistein, tyrphostin A25, and tyrphostin A47 blocked the volume-activated taurine efflux. These results suggest that the volume-activated taurine efflux in

K562 cells may not directly involve Ca2+, but may require the presence of calmodulin and/or PTK.
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 11 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:812257 HCAPLUS
DOCUMENT NUMBER: 136:320118
TITLE: Molecular cloning and sequence analyses of calcium/calmodulin-dependent protein kinase II from fetal and adult human brain. Sequence analyses of human brain calcium/calmodulin-dependent protein kinase II
AUTHOR(S): Li, Guangyu; Laabich, Aicha; Liu, L. Olivia; Xue, Jin; Cooper, Nigel G. F.
CORPORATE SOURCE: Department of Anatomical Sciences and Neurobiology, University of Louisville School of Medicine, Louisville, KY, 40202, USA
SOURCE: Molecular Biology Reports (2001), 28(1), 35-41
CODEN: MLBRBU; ISSN: 0301-4851
PUBLISHER: Kluwer Academic Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The aims of this study were to characterize specific mRNAs and the expression pattern for isoforms of calcium/calmodulin-dependent protein kinase II (CaMKII) in the human brain. We cloned and sequenced the CaMKII α and β subunit cDNAs, and used them to study the CaMKII expression in human brain. Four distinct isoforms of CAMKII were isolated. Two of them were characterized as CaMKII α and β subunits. The other two showed similar nucleotide sequences, but one had a 33-bp insertion relative to the α subunit, and the other had a 75-bp deletion relative to the β subunit. These alterations are located within the variable regions. These two isoforms were characterized as CaMKII αB and βe. Northern blot anal. showed that a 4.4-kb mRNA for the α isoform and a 3.9-kb mRNA for the β isoform were expressed in both human fetal and adult brain to different degrees. The results indicate that CaMKII expression is developmentally regulated. The CaMKII isoform expression was confirmed in human fetal and adult brain using RT-PCR with specific primers, which flanked the CaMKII variable regions. The CaMKII α, αB, β, β' and βe isoforms were characterized in both human fetal and adult brain.
REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 12 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:439719 BIOSIS
DOCUMENT NUMBER: PREV200000439719
TITLE: Determination of the genomic structure of calcium/calmodulin protein Kinase II gamma gene, a candidate gene for Type II diabetes.
AUTHOR(S): Gloyn, A. L. [Reprint author]; Hashim, Y. [Reprint author]; Ashcroft, S. J. H.
CORPORATE SOURCE: Diabetes Research Laboratories, Nuffield Department of Clinical Medicine, University of Oxford, Oxford, UK
SOURCE: Diabetologia, (August, 2000) Vol. 43, No. Supplement 1, pp. A82. print.
Meeting Info.: 36th Annual Meeting of the European Association for the Study of Diabetes. Jerusalem, Israel. September 17-21, 2000. European Association for the Study of Diabetes.
CODEN: DBTGAJ. ISSN: 0012-186X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English
ENTRY DATE: Entered STN: 18 Oct 2000
Last Updated on STN: 10 Jan 2002

L20 ANSWER 13 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:783640 HCAPLUS
DOCUMENT NUMBER: 130:150193
TITLE: Components of a calmodulin-dependent protein kinase cascade. Molecular cloning, functional characterization and cellular localization of Ca2+/calmodulin-dependent protein kinase kinase β
AUTHOR(S): Anderson, Kristin A.; Means, Raylene L.; Huang, Qi-Hui; Kemp, Bruce E.; Goldstein, Elaine G.; Selbert, Michele A.; Edelman, Arthur M.; Fremeau, Robert T.; Means, Anthony R.
CORPORATE SOURCE: Department of Pharmacology and Cancer Biology, Duke University, Durham, NC, 27710, USA
SOURCE: Journal of Biological Chemistry (1998), 273(48), 31880-31889
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Ca2+/calmodulin-dependent protein kinases I and IV (CaMKI and CaMKIV, resp.) require phosphorylation on an equivalent single Thr in the activation loop of subdomain VIII for maximal activity. Two distinct CaMKI/IV kinases, CaMKKα and CaMKKβ, were purified from rat brain and partially sequenced (Edelman, A. M., Mitchelhill, K., Selbert, M. A., Anderson, K. A., Hook, S. S., Stapleton, D., Goldstein, E. G., Means, A. R., and Kemp, B. E. (1996) J. Biol. Chemical 271, 10806-10810). We report here the cloning and sequencing of cDNAs for human and rat CaMKKβ, tissue and regional brain localization of CaMKKβ protein, and mRNA and functional characterization of recombinant CaMKKβ in vitro and in Jurkat T cells. The sequences of human and rat CaMKKβ demonstrate 65% identity and 80% similarity with CaMKKα and 30-40% identity with CaMKI and CaMKIV themselves. CaMKKβ is broadly distributed among rat tissues with highest levels in CaMKIV-expressing tissues such as brain, thymus, spleen, and testis. In brain, CaMKKβ tracks more closely with CaMKIV than does CaMKKα. Bacterially expressed CaMKKβ undergoes intramol. autophosphorylation, is regulated by Ca2+/CaM, and phosphorylates CaMKI and CaMKIV on Thr177 and Thr200, resp. CaMKKβ activates both CaMKI and CaMKIV when coexpressed in Jurkat T cells as judged by phosphorylated cAMP response element-binding protein-dependent reporter gene expression. CaMKKβ activity is enhanced by elevation of intracellular Ca2+, although substantial activity is observed at the resting Ca2+ concentration The strict Ca2+ requirement of CaMKIV-dependent phosphorylation of cAMP response element-binding protein, is therefore controlled at the level of CaMKIV rather than CaMKK.
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 14 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:550481 HCAPLUS
DOCUMENT NUMBER: 121:150481
TITLE: Roles of calmodulin-dependent protein kinase and phosphatase in calcium-dependent transcription of immediate early genes
AUTHOR(S): Enslen, Herve; Soderling, Thomas R.
CORPORATE SOURCE: Vollum Inst., Oregon Health Sci. Univ., Portland, OR, 97201, USA
SOURCE: Journal of Biological Chemistry (1994), 269(33), 20872-7
CODEN: JBCHA3; ISSN: 0021-9258

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recent studies indicate multiple mechanisms are involved in Ca2+ stimulation of gene expression. The authors have used cell-permeable, specific inhibitors of calmodulin-dependent protein kinases (CaM kinases) and phosphatase (calcineurin) to investigate the involvement of these enzymes in transcriptional regulation of three immediate early genes in PC12 cells stimulated with A23187 or KCl. Preincubation of PC12 cells with the CaM kinase inhibitor KN-62 blocked auto-phosphorylation of CaM kinase II in response to stimulation by the Ca2+ ionophore A23187. KN-62 treatment also resulted in a 60-70% inhibition of Ca2+-dependent transcription of c-fos, NGFI-A (zif 268), and NGFI-B (nur 77) as assessed by either Northern or nuclear run-on analyses. Preincubation with the calcineurin inhibitors FK-506 or cyclosporin A strongly enhanced expression of NGFI-A and blocked transcription of NGFI-B, but it had no significant effect on Ca2+-stimulated transcription of c-fos. Both FK-506 and KN-62 were specific for Ca2+-stimulated transcription as neither effected transcription in response to forskolin or phorbol ester (12-O-tetradecanoylphorbol-13-acetate) treatment. This is the first report of CaM kinase and calcineurin, in response to elevated intracellular Ca2+, would exert antagonistic effects on transcription of NGFI-A. Since inhibition of either the kinase or phosphatase decreased transcription of NGFI-B by 60-90%, this suggests that each enzyme is necessary but not sufficient for Ca2+ stimulation. These results indicate that CaM kinases and calcineurin can mediate broad and complex regulation of Ca2+-stimulated gene expression.

L20 ANSWER 15 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:406538 HCAPLUS
DOCUMENT NUMBER: 121:6538
TITLE: Calcium regulates processing of the Alzheimer amyloid protein precursor in a protein kinase C-independent manner
AUTHOR(S): Buxbaum, Joseph D.; Ruefli, Astrid A.; Parker, Carolyn A.; Cypess, Aaron M.; Greengard, Paul
CORPORATE SOURCE: Lab. Molecular Cellular Neuroscience, Rockefeller Univ., New York, NY, 10021, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1994), 91(10), 4489-93
CODEN: PNASA6; ISSN: 0027-8424
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Various first messengers linked to phospholipase C, including acetylcholine and interleukin 1, regulate the production both of the secreted form of the amyloid protein precursor (APP) and of amyloid β-protein. The authors have now identified intracellular signals which are responsible for mediating these effects. The authors show that activation of phospholipase C may affect APP processing by either of two pathways, one involving an increase in protein kinase C and the other an increase in cytoplasmic calcium levels. The effects of calcium on APP processing appear to be independent of protein kinase C activation. The observed effects of calcium on APP processing may be of therapeutic utility.

L20 ANSWER 16 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:262857 HCAPLUS
DOCUMENT NUMBER: 120:262857
TITLE: A Ca2+/calmodulin-dependent protein kinase, CaM kinase-Gr, expressed after transformation of primary human B lymphocytes by Epstein-Barr virus (EBV) is induced by the EBV oncogene LMP1
AUTHOR(S): Mosialos, George; Hanissian, Silva H.; Jawahar, Satya; Vara, Lisa; Kieff, Elliott; Chatila, Talal A.
CORPORATE SOURCE: Dep. Med., Harvard Med. Sch., Boston, MA, 02115, USA
SOURCE: Journal of Virology (1994), 68(3), 1697-705

CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB CaM kinase-Gr is a multifunctional Ca2+/calmodulin-dependent protein kinase which is enriched in neurons and T lymphocytes. The kinase is absent from primary human B lymphocytes but is expressed in Epstein-Barr virus (EBV)-transformed B-lymphoblastoid cell lines, suggesting that expression of the kinase can be up-regulated by an EBV gene product(s). The authors investigated the basis of CaM kinase-Gr expression in EBV-transformed cells and the mechanisms that regulate its activity therein by using an EBV-neg. Burkitt lymphoma cell line, BJAB, and BJAB cells converted to expression of individual EBV protein by single-gene transfer. CaM kinase-Gr expression was up-regulated in BJAB cells by EBV latent-infection membrane protein 1 (LMP1) but not by LMP2A or by nuclear proteins EBNA1, EBNA2, EBNA3A, and EBNA3C. In LMP1-converted BJAB cells, the kinase was functional and was dramatically activated upon crosslinking of surface IgM. Overlapping cDNA clones that encode human CaM kinase-Gr were sequenced, revealing 81% amino acid identity between the rat and human proteins. Transfection of BJAB cells with an expression construct for the human enzyme resulted in a functional kinase which was shown by epitope tagging to localize primarily to cytoplasmic and perinuclear structures. Induction of CaM kinase-Gr expression by LMP1 provides the first example of a Ca2+/calmodulin-dependent protein kinase up-regulated by a viral protein. In view of the key role played by LMP1 in B-lymphocyte immortalization by EBV, these findings implicate CaM kinase-Gr as a potential mediator of B-lymphocyte growth transformation.

L20 ANSWER 17 OF 68 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 94375404 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8089075
TITLE: cDNA cloning and expression of **human calmodulin**-dependent protein **kinase** IV.
AUTHOR: Kitani T; Okuno S; Fujisawa H
CORPORATE SOURCE: Department of Biochemistry, Asahikawa Medical College, Hokkaido.
SOURCE: Journal of biochemistry, (1994 Apr) 115 (4) 637-40. Journal code: 0376600. ISSN: 0021-924X.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D30742
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941031
Last Updated on STN: 19980206
Entered Medline: 19941018

AB Calmodulin-dependent protein kinase IV (CaM-kinase IV) is a Ca(2+)-responsive multifunctional protein kinase which occurs abundantly in the brain and thymus. A human cDNA clone encoding CaM-kinase IV was isolated from a Jurkat cell cDNA library and its nucleotide sequence was determined. The cDNA sequence encoded a protein consisting of 473 amino acids with a molecular weight of 51,925. The nucleotide sequence for the coding region and the deduced amino acid sequence showed 81 and 80% identities with those of the rat enzyme, respectively. Western blot analysis, using a polyclonal antibody raised against the recombinant human CaM-kinase IV, which was expressed in Escherichia coli, revealed two bands corresponding in mobility to molecular weights of 60,000 and 61,000, respectively, in a Jurkat cell extract. The antibody also cross-reacted with both isoforms of CaM-kinase IV from rat cerebellum, the apparent molecular weights being 62,000 and 64,000, respectively.

L20 ANSWER 18 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:261806 HCAPLUS
DOCUMENT NUMBER: 120:261806

TITLE: Ca2+-mobilizing agonists potentiate forskolin- and VIP-stimulated cAMP production in human colonic cell line, HT29-cI.19A: role of [Ca2+]i and protein kinase C
AUTHOR(S): Warhurst, G.; Fogg, K. E.; Higgs, N. B.; Tonge, A.; Grundy, J.
CORPORATE SOURCE: Epithelial Mem. Res. Cent., Univ. Manchester, Salford, UK
SOURCE: Cell Calcium (1994), 15(2), 162-74
CODEN: CECADV; ISSN: 0143-4160
DOCUMENT TYPE: Journal
LANGUAGE: English

AB This study examined the involvement of the Ca2+-signalling pathway in the regulation of agonist-stimulated cAMP responses in the human colonic adenocarcinoma cell line, HT29-cI.19A. The muscarinic agonist, carbachol (CCh) stimulated rapid increases in cellular IP3 and cytosolic Ca2+, [Ca2+]i in HT29-cI.19A cells. These were accompanied by an increase in basal cAMP levels and a marked (3-4-fold) potentiation of both forskolin- (FSK) and VIP-stimulated cAMP generation. Similar effects were observed with 2 other Ca2+-mobilizing agonists, neurotensin and ATP. The failure of CCh to elicit potentiation of adenylate cyclase in broken cell prepns. indicated an indirect action. Potentiation could be mimicked by the Ca ionophore, ionomycin, and thapsigargin and inhibited 70-90% by depleting intracellular Ca2+ stores suggesting that a rise [Ca2+]i is the primary mediator of this response. In contrast, increasing [Ca2+]i levels to > 500 nM inhibited FSK-stimulated cAMP generation. Addnl., protein kinase C (PKC) activators phorbol 12,13 dibutyrate (PDB) and 1-oleoyl-2-acetyl glycerol (OAG) potentiated FSK-stimulated cAMP production by 50-70% though PDB markedly inhibited the cAMP response to the receptor-mediated cAMP agonist, VIP. Neither effect could be elicited by the inactive phorbol ester, 4α-phorbol, 12,13 didecanoate (PDD). PKC inhibitors staurosporine and H7 reduced by ≈25% the CCh-induced potentiation of FSK-stimulated cAMP generation. These results suggest that stimulation of the phosphoinositidase C pathway in HT29-cI.19A colonocytes induces a sensitization of the adenylate cyclase system resulting in a dramatic amplification of agonist-stimulated cAMP generation. Increases in [Ca2+]i appear to be an important mediator of potentiation though activation of PKC may also play a significant role.

L20 ANSWER 19 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:537009 HCAPLUS
DOCUMENT NUMBER: 119:137009
TITLE: Expression of a calcium/calmodulin-dependent protein kinase, CaM kinase-Gr, in human T lymphocytes. Regulation of kinase activity by T cell receptor signaling
AUTHOR(S): Hanissian, Silva H.; Frangakis, Maria; Bland, Molly M.; Jawahar, Satya; Chatila, Talal A.
CORPORATE SOURCE: Div. Immunol., Child. Hosp., Boston, MA, 02115, USA
SOURCE: Journal of Biological Chemistry (1993), 268(27), 20055-63
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Ca2+/calmodulin-dependent protein kinase type Gr (CaM kinase-Gr) is a Ca2+/calmodulin-dependent protein kinase which is enriched in the brain and thymus. In this study, the authors examined the expression of CaM kinase-Gr in human lymphocytes and the regulation of its catalytic activity by antigen receptor signaling. CaM kinase-Gr was found selectively expressed in T lymphocytes in a developmentally regulated manner. It was present at severalfold higher levels in immature thymocytes (CD3low, CD4+CD8+) relative to mature thymocytes (CD3high, CD4+CD8-/CD8+/CD4-) or to circulating T lymphocytes. The kinase was preferentially expressed in CD4+ T lymphocytes, but was not detected in B

lymphocytes or in monocytes. The impact of T cell antigen receptor-CD3 complex (TCR·CD3) signaling on kinase activity was examined using Jurkat human leukemic T lymphocytes as a model. Treatment of Jurkat cells with anti TCR·CD3 monoclonal antibody induced rapid auto-phosphorylation of the kinase on serine residues and a dramatic, auto-phosphorylation-dependent enhancement of both Ca2+/calmodulin-dependent and autonomous kinase activity. Enzyme auto-phosphorylation and activation were dependent on the influx of extracellular Ca2+ following receptor signaling but could not be induced by an influx of extracellular Ca2+ triggered by ionophores, indicating that addnl. signals delivered via TCR·CD3 contribute to the activation of CaM kinase-Gr. These findings suggest a role for CaM kinase-Gr in T lymphocyte development and activation and indicate the presence of stringent regulatory mechanisms governing the activity of this kinase in situ.

L20 ANSWER 20 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:403901 HCAPLUS
DOCUMENT NUMBER: 119:3901
TITLE: Cloning and analysis of two new isoforms of multifunctional calcium/calmodulin-dependent protein kinase. Expression in multiple human tissues
AUTHOR(S): Nghiem, Paul; Saati, Shahin M.; Martens, Christine L.; Gardner, Phyllis; Schulman, Howard
CORPORATE SOURCE: Dep. Pharmacol., Stanford Med. Sch., Stanford, CA, 94305, USA
SOURCE: Journal of Biological Chemistry (1993), 268(8), 5471-9
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Multifunctional Ca2+/calmodulin-dependent protein kinase (CaM kinase) is a mediator of calcium signals in diverse signaling pathways. In human lymphocytes and epithelial tissues, CaM kinase activates a chloride channel via a Ca2+-dependent pathway which is preserved in cystic fibrosis. To characterize the CaM kinase present in these tissues the authors have cloned an isoform of this kinase from human T lymphocytes. The cDNA sequences of 2 variants of this human CaM kinase, γB and γC, are predicted to translate to 518 and 495 amino acids, resp. Amino acid differences between these isoforms and the rat brain γ isoform (which the authors refer to as γA) are localized to the variable domain. RNase protection of this variable region revealed the level of expression of γB and γC CaM kinase mRNAs in nine human tissues and cell lines. When transfected into Jurkat T cells, the γB cDNA encoded a functional kinase which cosedimented on sucrose gradients with endogenous T cell CaM kinase activity and formed a large multimeric enzyme. The recombinant γB isoform displayed 2 phases of autophosphorylation characteristic of CaM kinases, including the phase which converts it to a partially Ca2+-independent species. Site-directed mutagenesis of the predicted autoinhibitory domain yielded a mutant which was .apprx.37% active in the absence of Ca2+/calmodulin, confirming the region as critical for autoregulation, and suggesting this mutant as a tool for studying the role of CaM kinase in nonneuronal tissues.

L20 ANSWER 21 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:601647 HCAPLUS
DOCUMENT NUMBER: 119:201647
TITLE: Calcium-dependent protein kinase C activity of neutrophils in localized juvenile periodontitis
AUTHOR(S): Kurihara, Hidemi; Murayama, Yoji; Warbington, Martha L.; Champagne, Catherine M. E.; Van Dyke, Thomas E.
CORPORATE SOURCE: Dent. Sch., Okayama Univ., Okayama, 700, Japan
SOURCE: Infection and Immunity (1993), 61(8), 3137-42
CODEN: INFIBR; ISSN: 0019-9567
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Protein kinase C is a key mol. in neutrophil signal transduction after receptor stimulation by soluble bioactive mols. It has been reported that neutrophils from most patients with localized juvenile periodontitis (LJP) do not have a normal response after stimulation with a chemotactic ligand such as N-formyl-Met-Leu-Phe (fMLP). To further clarify the mechanism of this altered response and to confirm and expand earlier observations., the calcium-dependent protein kinase C activity of neutrophils from patients with LJP was evaluated. Peripheral blood neutrophils from patients and healthy subjects, age, sex, and race matched, were sonicated and subsequently subfractionated by ultracentrifugation into a soluble fraction (cytosol rich) and a particulate fraction (membrane rich). The calcium-dependent protein kinase C activity was evaluated in each fraction by phosphorylation of histone with radiolabeled ATP in the presence or in the absence of phorbol 12-myristate 13-acetate stimulation. Results revealed that the total calcium-dependent protein kinase C activity of neutrophils from patients with LJP and depressed chemotactic migration to fMLP (201.0 pmol/min/107 cells) was lower than that of neutrophils from healthy subjects (287.6 pmol/min/107 cells). The calcium-dependent protein kinase C activity in neutrophils from patients with LJP exhibited a pos. correlation with chemotactic migration to fMLP. The low activity of calcium-dependent protein kinase C in neutrophils from the patients reflected the low activity in the soluble fraction from the neutrophils. After stimulation with phorbol 12-myristate 13-acetate, the calcium-dependent protein kinase C activity was lower from patients with LJP than from healthy subjects. Thus, the lower calcium-dependent protein kinase C in neutrophils is a predisposing factor for LJP.

L20 ANSWER 22 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:188391 HCAPLUS
DOCUMENT NUMBER: 120:188391
TITLE: Analysis of calcium-dependent protein kinase-C isoenzymes in intrinsically resistant cloned lines of LoVo cells: reversal of resistance by kinase inhibitor 1-(5-isoqinolinylsulfonyl) 2-methylpiperazine
AUTHOR(S): Dolfini, Ersilia; Dasdia, Teresa; Perletti, Gianpaolo; Romagnoni, Marilena; Piccinini, francesco
CORPORATE SOURCE: Inst. Pharmacol., Univ. Milan, Milan, 20129, Italy
SOURCE: Anticancer Research (1993), 13(4), 1123-7
CODEN: ANTRD4; ISSN: 0250-7005
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In order to investigate the involvement of Protein Kinase C (PKC) in the signal transduction mechanisms related to intrinsic chemoresistance, 2 cellular clones were isolated from LoVo/WT colon adenocarcinoma cell line and their cytogenetic pattern was studied: LoVo C1.7 was intrinsically resistant to Doxorubicin while LoVo C1.5 showed the same resistance index as the mixed parental cell population. Two PKC isoforms, immunol. identified as β and αPKC, were isolated from the cytosolic fraction of all cell types and one single peak of αPKC was obtained from the particulate fraction. Resistant LoVo C1.7 cells showed a significant increase of PKC activity; preincubation with H-7 induced PKC inhibition and reversal of drug resistance. These data suggest that in the authors' cell system the identified calcium-dependent PKC subtypes can play a role in the mechanisms of intrinsic resistance.

L20 ANSWER 23 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:98813 HCAPLUS
DOCUMENT NUMBER: 118:98813
TITLE: Synergy between calcium and protein kinase C is the major factor in determining the level of secretion from human platelets
AUTHOR(S): Walker, Trevor R.; Watson, Steve P.
CORPORATE SOURCE: Dep. Pharmacol., Univ. Oxford, Oxford, OX1 3QT, UK
SOURCE: Biochemical Journal (1993), 289(1), 277-82

CODEN: BIJOAK; ISSN: 0306-3275
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The aim of this study was to establish further the role of protein kinase C in aggregation and secretion of 5-HT from human platelets by using the selective inhibitor Ro 31-8220. Ro 31-8220 (3 μM) inhibited completely phosphorylation of pleckstrin, the major protein kinase C substrate, induced by thrombin, A 23187 or phorbol dibutyrate (PDBu). Myosin light-chain phosphorylation induced by PDBu was also inhibited completely, but that induced by thrombin or A 23187 was only inhibited partially. As myosin light chain is a substrate for both myosin light-chain kinase and protein kinase C, these results suggest that Ro 31-8220 is inhibiting only the protein kinase C-induced phosphorylation and that Ro 31-8220 has a greater selectivity to protein kinase C than does its structural analog staurosporine. The stimulation of secretion of 5-HT by maximally effective concns. of thrombin and A 23187 was decreased significantly by 3 μM Ro 31-8220, but not inhibited completely. These results indicate a major role for protein kinase C in the stimulation of secretion by agonist- and ionophore-induced activation. On its own, a maximal concentration of PDBu induced a small degree of secretion (3.3%), but potentiated markedly the response to a submaximal concentration of A 23187 (300 nM) to a level greater than seen with a maximal concentration of A 23187. A similar set of results was also seen with aggregation, but not with shape change. Apparently, the signaling event for secretion and aggregation is Ca2+, and this is potentiated markedly by protein kinase C. In the case of secretion, it appears that it is the synergy which is the major determining factor in influencing the extent of secretion.

L20 ANSWER 24 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:19839 HCAPLUS
DOCUMENT NUMBER: 118:19839
TITLE: Possible involvement of protein kinase C and calcium in GSH efflux from Hep G2 cells
AUTHOR(S): Sato, Chifumi; Liu, Jin Hong; Tang, Liang; Sakai, Yoshinori; Yauchi, Tsunehito; Izumi, Namiki; Liu, Jian; Takano, Takehito; Marumo, Fumiaki
CORPORATE SOURCE: Fac. Med., Tokyo Med. Dent. Univ., Tokyo, 113, Japan
SOURCE: Life Sciences (1992), 51(26), 2057-63
CODEN: LIFSAK; ISSN: 0024-3205
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of protein kinase C modulations and Ca mobilization on GSH efflux in Hep G2 cells were investigated. GSH efflux from Hep G2 cells was increased by a phorbol ester. Staurosporine, an inhibitor of protein kinase C, diminished phorbol ester-stimulated GSH efflux from the cells. GSH efflux was neg. correlated with extracellular Ca concns. Verapamil enhanced GSH efflux, whereas ATP decreased GSH efflux. The latter effect was diminished in the absence of extracellular Ca. Protein kinase C and Ca mobilization may be crucial factors in GSH efflux from human hepatocytes.

L20 ANSWER 25 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:4523 HCAPLUS
DOCUMENT NUMBER: 118:4523
TITLE: Differential megakaryocytic desensitization to platelet agonists
AUTHOR(S): Dorn, Gerald W., II; Davis, Michael G.
CORPORATE SOURCE: Coll. Med., Univ. Cincinnati, Cincinnati, OH, 45267, USA
SOURCE: American Journal of Physiology (1992), 263(4, Pt. 1), C864-C872
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Platelets are released into the peripheral circulation from the bone marrow where they arise as fragments of megakaryocyte cytoplasm. To characterize the effects of platelet agonists on megakaryocytes, the authors examined Ca2+ signaling and desensitization to thrombin, the TxA2 mimetic U46619, and platelet-activating factor (PAF) in cultured CHRF-288-11 megakaryocytic cells. Initially, the authors compared agonist-stimulated Ca2+ transients in fura-2-loaded CHRF-288-11 cells and human platelets. The 50% effective concentration values for the agonists to increase free cytosolic Ca2+ were as follows: thrombin (0.11 U/mL in CHRF, 0.19 U/mL in platelets), U46619 (147 nM in CHRF, 157 nM in platelets), and PAF (15 nM in CHRF, 16 nM in platelets). CHRF-288-11 thrombin, TxA2, and PAF receptors were demonstrated to be coupled to phospholipase C because each of the agonists stimulated phosphatidylinositol hydrolysis in myo-[3H]inositol-loaded CHRF-288-11 cells and pharmacol. inhibition of phospholipase C blunted agonist-stimulated Ca2+ signaling. CHRF-288-11 cells exposed to the 3 agonists for 1 h showed different patterns and extent of homologous and heterologous desensitization. Protein kinase C activation appeared to be necessary but not sufficient for desensitization because 1) activation of protein kinase C with phorbol 12-myristate 13-acetate inhibited the Ca2+ responses to all 3 agonists, 2) inhibition of protein kinase C with staurosporine attenuated subsequent desensitization to each agonist, and 3) each agonist increased protein kinase C activity in CHRF-288-11 cell homogenates.

L20 ANSWER 26 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:588936 HCAPLUS
DOCUMENT NUMBER: 117:188936
TITLE: The roles of protein kinase C and intracellular calcium in the secretion of von Willebrand factor from human vascular endothelial cells
AUTHOR(S): Carew, Mark A.; Paleolog, Ewa M.; Pearson, Jeremy D.
CORPORATE SOURCE: Clin. Res. Cent., MRC, Harrow/Middx., HA1 3UJ, UK
SOURCE: Biochemical Journal (1992), 286(2), 631-5
CODEN: BIJOAK; ISSN: 0306-3275
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Secretion of von Willebrand factor (vWf) from storage granules in human umbilical-vein endothelial cells was studied in vitro. Either elevation of intracellular Ca2+ concentration ([Ca2+]i) with a Ca2+ ionophore or activation of protein kinase (PK) C by phorbol 12-myristate 13-acetate caused vWf secretion, and together the agents acted synergistically. However, when vWf release was stimulated by receptor-mediated agonists, selective inhibition of PKC had no effect on histamine-induced secretion and significantly elevated thrombin-induced secretion. Furthermore, ATP, which efficiently elevates [Ca2+]i in these cells, was a very poor effector of vWf release. Apparently, elevation of [Ca2+]i by physiol. agonists is necessary for vWf release, but other signalling mechanisms, as yet uncharacterized, but not due to PKC activation, are required for full induction of the secretory pathway.

L20 ANSWER 27 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:5127 HCAPLUS
DOCUMENT NUMBER: 118:5127
TITLE: Neutrophil signal transduction: calcium, kinases, and fusion
AUTHOR(S): Smolen, James E.
CORPORATE SOURCE: Med. Sch., Univ. Michigan, Ann Arbor, MI, 48109-0684, USA
SOURCE: Journal of Laboratory and Clinical Medicine (1992), 120(4), 527-32
CODEN: JLCMAK; ISSN: 0022-2143
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review, with 61 refs., of various factors involved in stimulus-response coupling in human neutrophils (receptors, G proteins, phospholipases, Ca2+, and protein kinases), as well as factors involved in fusion of the neutrophil granule membranes with the phagosome or plasma membranes with the accompanying discharge of granule contents (these factors include Ca2+, annexins, and lipids). Methods used for assaying and studying fusion and their disadvantages as well as the specificity and presence of high false-neg. rates for contents-mixing assays, are discussed.

L20 ANSWER 28 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:407160 HCAPLUS
DOCUMENT NUMBER: 119:7160
TITLE: The role of calcium and protein kinase C in the intracellular killing of bacteria by human monocytes
AUTHOR(S): Zheng, L.; Nibbering, P. H.; Van Furth, R.
CORPORATE SOURCE: Dep. Infect. Dis., Univ. Hosp., Leiden, 2300 RC, Neth.
SOURCE: Mononucl. Phagocytes (1992), 483-8. Editor(s): Van Furth, Ralph. Kluwer: Dordrecht, Neth. CODEN: 59AEA4
DOCUMENT TYPE: Conference
LANGUAGE: English
AB The role is reported here of the second messengers cytosolic free Ca2+ and protein kinase C in the intracellular killing of bacteria (Staphylococcus aureus) by human monocytes.

L20 ANSWER 29 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:446149 HCAPLUS
DOCUMENT NUMBER: 117:46149
TITLE: Anti-class II MHC antibody induces multinucleated giant cell formation from peripheral blood monocytes
AUTHOR(S): Orentas, Rimas J.; Reinlib, Leslie; Hildreth, James E. K.
CORPORATE SOURCE: Sch. Med., Johns Hopkins Univ., Baltimore, MD, 21205, USA
SOURCE: Journal of Leukocyte Biology (1992), 51(3), 199-209 CODEN: JLBIE7; ISSN: 0741-5400
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Multinucleated giant cells (MGCs) are an integral part of the host immune response to infectious disease and are seen in granulomas induced by pathogens and inorg. substances. Peripheral blood monocytes, when cultured in the presence of anti-class II major histocompatibility complex monoclonal antibodies (MHC mAbs) and lymphocyte-conditioned medium form MGCs within 48 h. MGC formation was strictly dependent on the presence of anti-class H MHC mAbs and lymphocyte-conditioned medium. MGC formation was not induced by mAbs to other monocyte surface proteins. None of the previously identified macrophage fusion factors (calcitriol, interleukin 4, interferon-γ) were able to substitute for the lymphocyte-conditioned medium in the assay; however, the conditioned medium could be replaced by phorbol 12-myristate 13-acetate. Also, the induction of MGCs by anti-class II MHC antibody and phorbol ester requires protein kinase C activity. In analyzing the signal induced by anti-class II MHC mAbs, crosslinking of the class II MHC antigens with intact mAbs, or with F(ab')2 fragments of anti-class II MHC mAbs and F(ab')2 fragments of rabbit antimouse (RAM) IgG, produced an intracellular Ca rise. Furthermore, Ca channel activity is necessary for MGC formation. Apparently, MGC formation is a tightly regulated differentiative pathway of peripheral blood monocytes that is dependent on protein kinase C second messenger systems and involves an increase in intracellular Ca concentration

L20 ANSWER 30 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:649192 HCAPLUS
DOCUMENT NUMBER: 117:249192
TITLE: Mechanisms of activation of sodium/hydrogen ion

exchange in human osteoblast-like SaOS-2 cells
AUTHOR(S): Graham, Camilla S.; Tashjian, Armen H., Jr.
CORPORATE SOURCE: Dep. Mol. Cell. Toxicol., Harvard Sch. Public Health, Boston, MA, 02115, USA
SOURCE: Biochemical Journal (1992), 288(1), 137-43
CODEN: BIJOAK; ISSN: 0306-3275
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Because of the importance of pH homeostasis in bone and the current uncertainty about the mechanisms by which intracellular pH (pHi) is regulated in this tissue, the authors have investigated the roles of cytosolic free Ca2+ concns. ([Ca2+]i) and protein kinase C on the activation of Na+/H+ exchange in human osteoblast-like SaOS-2 cells. [Ca2+]i and pHi were measured using Fura-2 and BCECF, resp. The basal pHi in HCO3--free buffer was 7.36 units. Addition of ionomycin in Ca2+-containing buffer did not cause a rise in basal pHi; however, addition of PMA did cause a slowly developing rise in resting pHi of 0.14 unit over 4-5 min. Nigericin, a K+/H+ ionophore, caused an abrupt fall in pHi to 6.70 units. In nigericin-pretreated cells, PMA caused a rapid rise in pHi without changing the [Ca2+]i. In acidified cells, ionomycin increased [Ca2+]i and pHi in a parallel concentration-dependent (30-500 nM) manner. This action of ionomycin occurred in both the presence and the nominal absence of extracellular Ca2+. Ionomycin-induced alkalinization depended on extracellular Na+ and was inhibited in cells incubated with hexamethylene amiloride. When the incremental increase in [Ca2+]i induced by ionomycin was blocked by preincubation with BAPTA/AM, the effect on pHi was inhibited. Staurosporine, a protein kinase C inhibitor, blocked the action of PMA on pHi, but it had no effect on the ionomycin-induced increase in pHi. The action of ionomycin was not due to osmotic shock. Apparently, SaOS-2 cells have a protein kinase C-activatable Na+/H+ antiporter that is also stimulated, in acidified cells, in a concentration-dependent fashion by transients in [Ca2+]i which act via a non-protein kinase C pathway.

L20 ANSWER 31 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1992:531062 BIOSIS
DOCUMENT NUMBER: PREV199243116762; BR43:116762
TITLE: G PROTEIN-REGULATED PHOSPHOLIPASES C D AND A-2-MEDIATED SIGNALLING IN NEUTROPHILS.
AUTHOR(S): COCKCROFT S [Reprint author]
CORPORATE SOURCE: DEP PHYSIOL, ROCKEFELLER BUILDING, UNIV COLL LONDON, UNIVERSITY ST, LONDON WC1E 6JJ, UK
SOURCE: Biochimica et Biophysica Acta, (1992) Vol. 1113, No. 2, pp. 135-160.
CODEN: BBACAQ. ISSN: 0006-3002.
DOCUMENT TYPE: Article
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 26 Nov 1992
Last Updated on STN: 24 Dec 1992

L20 ANSWER 32 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:610496 HCAPLUS
DOCUMENT NUMBER: 117:210496
TITLE: Effect of immunological and pharmacologic agents on acetylcholine receptor clusters of skeletal muscle
AUTHOR(S): Kojima, Hisanori
CORPORATE SOURCE: Sch. Med., Kanazawa Univ., Kanazawa, 920, Japan
SOURCE: Kanazawa Daigaku Juzen Igakkai Zasshi (1992), 101(1), 57-67
CODEN: JUZIAG; ISSN: 0022-7226
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB To gain insight into how the acetylcholine receptor (AChR) is dispersed by

antigen-specific humoral factor in myasthenia gravis, the effects of myasthenic patient serum or pharmacol. agents on AchR clusters were investigated. At first, the cultured rat myotubes were stained with fluorescein isothiocyanate conjugated α-bungarotoxin (FITC-α-BuTx) and changes caused by the serum or drugs were determined by the use of laser cytometer (ACAS 570). Serum samples from 17 myasthenia gravis (MG) patients caused a dispersal of AChR clusters in quant. correlation with anti-AChR antibody titers. The fluorescent patterns (area or average of fluorescence intensity) of AChR clusters remaining after the treatment with the serum samples showed no difference between the patient group and the control group. FRAP (fluorescence recovery after photobleaching) anal. also showed no effect of myasthenic sera on the myotube membrane fluidity. Calmodulin antagonist (W-7) and protein kinase activator C (TPA) induced a loss of AChR clusters. On the other hand, colchicine, cytochalasin B, and drugs effective on intracellular cAMP concentration (dibutyryl cAMP, CGRP) did not disrupt or induce the formation of AChR clusters. These results, obtained by the use of clin. and pharmacol. samples, lead to the possibility that the myasthenic antibody may influence the interaction between AChR and the submembranous system, and the submembranous system is stabilized by calmodulin and interfered with by protein kinase C.

L20 ANSWER 33 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1992:200180 BIOSIS
DOCUMENT NUMBER: PREV199242093255; BR42:93255
TITLE: INCREASED PHOSPHORYLATION OF ELONGATION FACTOR 2 IS RESPONSIBLE FOR REDUCED POLYSOME ACTIVITY IN ALZHEIMER'S DISEASE BRAINS.
AUTHOR(S): JOHNSON G [Reprint author]; MERRIL C; BIERER L; HAROUTUNIAN V; SUGAR J; WALLACE W
CORPORATE SOURCE: LAB BIOCHEM GENETICS, NIMH, WASHINGTON, DC, USA
SOURCE: Society for Neuroscience Abstracts, (1991) Vol. 17, No. 1-2, pp. 726.
Meeting Info.: 21ST ANNUAL MEETING OF THE SOCIETY FOR NEUROSCIENCE, NEW ORLEANS, LOUISIANA, USA, NOVEMBER 10-15, 1991. SOC NEUROSCI ABSTR.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 16 Apr 1992
Last Updated on STN: 2 May 1992

L20 ANSWER 34 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1991:448800 BIOSIS
DOCUMENT NUMBER: PREV199141086535; BR41:86535
TITLE: NEURONAL MUSCARINIC RECEPTORS AND PHOSPHOINOSITIDE METABOLISM.
AUTHOR(S): NAAHORSKI S R [Reprint author]; LAMBERT D G; WOJCIKIEWICZ R J H; CHALLISS R A J; SAFRANY S T
CORPORATE SOURCE: DEP PHARMACOL AND THERAPEUTICS, UNIV LEICESTER, PO BOX 138, MEDICAL SCIENCES BUILD, UNIVERSITY RD, LEICESTER LE1 9HN, UK
SOURCE: Biochemical Society Transactions, (1991) Vol. 19, No. 2, pp. 416-422.
Meeting Info.: 637TH MEETING OF THE BIOCHEMICAL SOCIETY, BIRMINGHAM, ENGLAND, UK, DECEMBER 18-20, 1990. BIOCHEM SOC TRANS.
CODEN: BCSTB5. ISSN: 0300-5127.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 8 Oct 1991
Last Updated on STN: 8 Oct 1991

L20 ANSWER 35 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1991:485667 HCAPLUS
DOCUMENT NUMBER: 115:85667
TITLE: Down-regulation of messenger ribonucleic acid and protein levels for estrogen receptors by phorbol ester and calcium in MCF-7 cells
AUTHOR(S): Ree, A. H.; Landmark, B. F.; Walaas, S. I.; Lahooti, H.; Eikvar, L.; Eskild, W.; Hansson, V.
CORPORATE SOURCE: Inst. Med. Biochem., Univ. Oslo, Oslo, 0317, Norway
SOURCE: Endocrinology (1991), 129(1), 339-44
CODEN: ENDOAO; ISSN: 0013-7227
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Treatment of MCF-7 cells, a human mammary carcinoma cell line, with phorbol ester [12-O-tetradecanoylphorbol-13-acetate (TPA)] or calcium ionophore (A23187) was associated with striking effects on levels of estrogen receptor (ER) mRNA, specific binding of 17β-[3H]estradiol ([3H]E2), and immunoreactive ER. TPA (10-7 M) caused a time-dependent reduction of ER mRNA which was below the level of detection after 9 h. Similar effects of TPA appeared on levels of specific binding of [3H]E2 as well as immunoreactive ER. In contrast, TPA induced an increase in mRNA for β-actin. Incubation of MCF-7 cells with increasing concns. of TPA (10-11-10-7 M) was associated with biphasic effects on ER mRNA and proteins. Levels of immunoreactive progesterone receptors (PR) were induced by E2 (10-9 M) in a time-dependent manner. In the presence of TPA (10-7 M), where ER levels were suppressed, no induction of PR was observed Removal of TPA (10-7 M) after 10 h (ER mRNA) or 22 h (ER proteins) of treatment was associated with a continued suppression of both mRNA and protein levels during the entire incubation period (48 h). Treatment with A23187 (2 + 10-7 M) also caused a time-dependent down-regulation of levels of ER mRNA and proteins. These effects occurred somewhat more slowly than those of TPA. Levels of β-actin mRNA were not changed by this treatment. These results indicate that changes in estrogen sensitivity are mediated by calcium-dependent protein kinases in human mammary carcinoma MCF-7 cells.

L20 ANSWER 36 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1991:604340 HCAPLUS
DOCUMENT NUMBER: 115:204340
TITLE: Platelet activation by diacylglycerol or ionomycin is inhibited by nitroprusside
AUTHOR(S): Doni, Maria Gabriella; Deana, Renzo; Padoin, Emilia; Ruzzene, Maria; Alexandre, Adolfo
CORPORATE SOURCE: Fac. Med. Surg., Univ. Padova, Padua, 35121, Italy
SOURCE: Biochimica et Biophysica Acta (1991), 1094(3), 323-9
CODEN: BBACAQ; ISSN: 0006-3002
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Expts. were performed to elucidate the role of cGMP on platelet activation induced by protein kinase C (PKC) activators and calcium ionophore. Human platelets were pretreated with acetylsalicylic acid and with hirudin and apyrase. Aggregation and ATP secretion in response to the PKC activators PMA and 1-oleoyl-2-acetylglycerol (OAG) were inhibited by the nitrovasdilator sodium nitroprusside (SNP), an activator of guanylate cyclase, and by 8-Br-cGMP. The expts. were performed in the presence of M & B 22948, an inhibitor of cGMP phosphodiesterase. SNP and 8-Br-cGMP also inhibited platelet aggregation and secretion evoked by the ionophore ionomycin. In fura-2 loaded platelets SNP did not affect basal cytosolic Ca2+ level nor the rise induced by low concns. of ionomycin, both in the presence and absence of extracellular Ca2+. The phosphorylation of the 47- and 20-kDa proteins induced by ionomycin or PMA were not decreased by SNP or 8-Br-cGMP. The present results suggest that cGMP is able to inhibit both the PKC and the Ca2+-dependent pathways leading to platelet

activation by interfering, similarly to cAMP, with processes following protein phosphorylation, close to the effector systems.

L20 ANSWER 37 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1991:140399 HCAPLUS
DOCUMENT NUMBER: 114:140399
TITLE: WS-1 human fibroblasts contain distinct calcium and protein kinase C-mediated pathways for activation of sodium/hydrogen ion exchange: contracting effects of thrombin and PMA
AUTHOR(S): Hendey, Bill; Mamrack, Mark D.
CORPORATE SOURCE: Dep. Biol. Sci., Wright State Univ., Dayton, OH, 45435, USA
SOURCE: Journal of Cellular Physiology (1991), 146(2), 290-7
CODEN: JCLLAX; ISSN: 0021-9541
DOCUMENT TYPE: Journal
LANGUAGE: English
AB PMA and thrombin were examined for their ability to activate Na+/H+ exchange in growth-arrested WS-1 human fibroblasts. PMA or thrombin caused a cytoplasmic alkalinization that required extracellular Na+ and was sensitive to 1 mM amiloride, suggesting that the rise in pH was mediated by the Na+/H+ exchanger. However, PMA and thrombin activated Na+/H+ exchange by distinctly different mechanisms. The rate of cytoplasmic alkalinization caused by 30 nM PMA was slower than 10 nM thrombin. The PMA-induced pH change was sensitive to the protein kinase inhibitors staurosporine (50 nM) and H 7 (100 μM). No increase in intracellular Ca2+ was observed after PMA treatment and the cytoplasmic alkalinization caused by PMA was not sensitive to the drug TMB 8 (200 μM) or the intracellular Ca2+ chelator BAPTA. In contrast, the thrombin-induced rise in cytoplasmic pH was sensitive to 50 nM staurosporine and only partially reduced with 100 μM H 7. The thrombin-induced activation of Na+/H+ exchange was inhibited by 200 μM TMB 8 or pretreatment with BAPTA. PMA caused translocation of PKC activity from a cytoplasmic to membrane fraction whereas thrombin did not. Pretreatment with 50 nM staurosporine significantly reduced measurable PKC activity with or without PMA treatment. PMA and thrombin were also examined for their ability to induce DNA synthesis in growth-arrested WS-1 human fibroblasts. Unlike thrombin, PMA did not stimulate [3H]thymidine incorporation in cells serum-deprived for 48 h. In addition, PMA inhibited thrombin-induced DNA synthesis when added at the same time or as late as 10 h after thrombin addition Therefore, thrombin and PMA activate Na+/H+ exchange by distinct pathways, but only the thrombin-induced pathway correlates with a mitogenic response.

L20 ANSWER 38 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1991:406793 HCAPLUS
DOCUMENT NUMBER: 115:6793
TITLE: Mechanism of lymphocyte abnormality associated with HLA-B8/DR3 in clinically healthy individuals. Cellular basis of impaired lymphocyte responsiveness To calcium ionophore and phorbol ester
AUTHOR(S): Hashimoto, Shu; Sawada, Shigemasa; Horie, Takashi
CORPORATE SOURCE: Sch. Med., Nihon Univ., Tokyo, 173, Japan
SOURCE: Nichidai Igaku Zasshi (1991), 50(2), 128-30
CODEN: NICHAS; ISSN: 0029-0424
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Highly purified T cells were stimulated with Ca ionophore and phorbol ester in an attempt to examine the mechanism of the decreased lymphocyte responsiveness. No significant difference in proliferative response of highly purified T cells to Ca ionophore and phorbol ester was observed between healthy individuals with and without HLA-B8/DR3, suggesting no abnormal function involved in increased protein kinase C and Ca2+ in T cell.

L20 ANSWER 39 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:841 HCAPLUS
DOCUMENT NUMBER: 116:841
TITLE: Control of steroidogenesis by the calcium messenger system in human adrenocortical cells
AUTHOR(S): Laird, S. M.; Hinson, J. P.; Vinson, G. P.; Mallick, N.; Kapas, S.; Teja, R.
CORPORATE SOURCE: Med. Coll., St. Bartholomew's Hosp., London, EC1M 6BQ, UK
SOURCE: Journal of Molecular Endocrinology (1991), 6(1), 45-51
CODEN: JMLEEI; ISSN: 0952-5041
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The involvement of the Ca messenger system in the control of steroidogenesis in the rat and bovine adrenal cortex has been studied extensively. However the role of these 2nd messengers in the control of human adrenocortical function is not established. This was therefore studied by incubating collagenase-dispersed human adrenocortical cells with the Ca ionophore A 23187 and the protein kinase C activator TPA. The effects of the Ca channel blocker verapamil on basal and stimulated steroidogenesis were also studied. Both TPA (1 pmol/1-10 μmol/L) and A 23187 (1 nmol/1-10 μmol/L) caused a dose-dependent increase in cortisol, aldosterone, and corticosterone production Verapamil (10 μmol/L) inhibited the increase in aldosterone, corticosterone, and cortisol produced in response to ACTH(1-24), K, and desacetyl-αMSH. Unlike previous results in the rat, these effects were not specific for aldosterone secretion. The results suggest that, as in other species, Ca mobilization and protein kinase C activation have a role in the control of steroidogenesis in the human adrenal cortex. However, in contrast to the rat, these mechanisms appear to be involved in the control of steroidogenesis in both the zona glomerulosa and inner zone cells.

L20 ANSWER 40 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1990:133108 HCAPLUS
DOCUMENT NUMBER: 112:133108
TITLE: Regulation of insulin-like growth factor I gene expression in the human macrophage-like cell line U937
AUTHOR(S): Nagaoka, I.; Trapnell, B. C.; Crystal, R. G.
CORPORATE SOURCE: Pulm. Branch, Natl. Heart, Lung, Blood Inst., Bethesda, MD, 20892, USA
SOURCE: Journal of Clinical Investigation (1990), 85(2), 448-55
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Activated macrophages release tissue forms of insulin-like growth factor I (IGF-I), 20-25-kilodalton products of the IGF-I gene, thus providing an extracellular growth and differentiation signal at sites of inflammation. To examine the control of IGF-I gene expression in mononuclear phagocytes, the human macrophage like cell line U937 was evaluated at rest and after surface activation with phorbol myristate acetate (PMA) or Ca2+ ionophore. Northern anal. and RNAse protection anal. with 32P-labeled IGF-I-specific probes demonstrated that the IGF-I mRNA transcripts of resting U937 cells were similar in size and amount to those of resting human alveolar macrophages, mononuclear phagocytes known to express the IGF-I gene. Nuclear run-off assays demonstrated that surface activation of U937 cells increased the transcription rate of the IGF-I gene 4-5-fold, a process that was inhibited by cycloheximide, suggesting that active protein synthesis was involved in the activation pathway. Despite this, cytoplasmic IGF-I mRNA levels after surface activation declined markedly, a process blocked by a protein kinase C inhibitor (for PMA activation) or a calmodulin antagonist (for Ca2+ ionophore activation). Like the increased transcription of the IGF-I gene, modulation of IGF-I mRNA transcript levels required active protein synthesis; in the presence of

cycloheximide constitutive IGF-I mRNA levels increased and surface activation no longer caused a decrease in transcript number Interestingly, surface activation caused a rapid release of IGF-I, even in the presence of a protein synthesis inhibitor, suggesting that mononuclear phagocytes have a preformed, stored, releasable pool of IGF-I. Together these observations demonstrate that IGF-I gene expression is complex and probably involves control of transcription rate, cytoplasmic mRNA levels possibly mediated through protein kinase C, Ca influx and calmodulin, and finally, release of preformed IGF-I from a storage pool.

L20 ANSWER 41 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1990:512889 HCAPLUS
DOCUMENT NUMBER: 113:112889
TITLE: Calcium-stimulatable and protein kinase C-inhibitable accumulation of inositol 1,3,4,6-tetrakisphosphate in human platelets
AUTHOR(S): King, Warren G.; Downes, C. Peter; Prestwich, Glenn D.; Rittenhouse, Susan E.
CORPORATE SOURCE: Coll. Med., Univ. Vermont, Burlington, VT, 05405, USA
SOURCE: Biochemical Journal (1990), 270(1), 125-31
CODEN: BIJOAK; ISSN: 0306-3275
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Thrombin-stimulated (10 s) human platelets produced Ins(1,4,5)P3 and an addnl. inositol trisphosphate (InsP3), in approx. a 1:20 ratio. The major InsP3 co-migrates with Ins(1,3,4)P3 on strong-anion-exchange HPLC. To identify this species unequivocally, putative Ins(1,3,4)P3 obtained from thrombin-stimulated myo-[3H]inositol-labeled platelets was treated with NaIO4/NaBH4 or 4-phosphomonoesterase. The products indicate that the major InsP3 is at least 90% D-Ins(1,3,4)P3. D-[3H]Ins(1,3,4)P3 added to saponin-permeabilized platelets is hydrolyzed to an InsP2 (7.8%) and phosphorylated by kinase to yield an inositol polyphosphate (0.9%) in 5 min. The phosphorylation product co-migrates with Ins(1,3,4,6)P4 on Partisphere WAX HPLC. Under similar conditions, L-[3H]Ins(1,3,4)P3 is dephosphorylated but not phosphorylated. Relative phosphatase:kinase ratios are 8.7:1 (Vmax. values) and 0.86:1 (Km values) with respect to D-Ins(1,3,4)P3. The kinase activity is predominantly cytosolic (96.8% of total activity) in freeze-thaw-disrupted platelets, and the accumulation of its product is Ca2+-dependent. The activity is identified as a 6-kinase on the basis of its product's insensitivity to 5-phosphomonoesterase, resistance to periodate oxidation and co-migration with standard Ins(1,3,4,6)P4 on HPLC. Incubation of platelets with β-phorbol dibutyrate (β-PDBu, 76 nM), causing activation of protein kinase C, results in a 57.5% inhibition (reversibly by the protein kinase C inhibitor staurosporine) of Ins(1,3,4,6)P4 accumulation. α-PDBu, which does not stimulate protein kinase C, has no effect. Stimulation of intact platelets with thrombin results in the production of Ins(1,3,4,6)P4 (1.4-fold rise in 30 s) and Ins(1,3,4,5)P4, with the latter being the major InsP4 species. Accumulation of Ins(1,3,4,6)P4 is slightly delayed in comparison with Ins(1,3,4)P3 and is relatively small. Evidently, the major route of Ins(1,3,4)P3 metabolism in stimulated human platelets is via phosphatase action.

L20 ANSWER 42 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1989:205660 HCAPLUS
DOCUMENT NUMBER: 110:205660
TITLE: Calmodulin and protein kinase C antagonists also inhibit the calcium-dependent protein protease calpain I
AUTHOR(S): Brumley, Lynn M.; Wallace, Robert W.
CORPORATE SOURCE: Dep. Pharmacol., Univ. Alabama, Birmingham, AL, 35294, USA
SOURCE: Biochemical and Biophysical Research Communications (1989), 159(3), 1297-303

CODEN: BBRCA9; ISSN: 0006-291X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The calmodulin and protein kinase C antagonists melittin, calmidazolium, N-(6-aminohexyl)-5-chloro-1-naphthalenesulfonamide (W7), and trifluoperazine (TFP) also inhibit the activity of the human erythrocyte Ca2+-dependent protease calpain I. W-5, the nonchlorinated derivative of W-7, was ineffective as an inhibitor of calpain I, just as it is for calmodulin and protein kinase C. Concentration-response studies provided the following IC50
values: melittin, 2.9 μM; calmidazolium, 6.2 μM; trifluoperazine, 130 μM; W-7, 251 μM. These values indicate that the compds. have affinities 10-600-fold less for calpain I than for calmodulin; however, the affinities of the inhibitory compds. are comparable for calpain I and protein kinase C. Kinetic anal. indicates that the compds. are competitive inhibitors of calpain I with respect to substrate.

L20 ANSWER 43 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1989:324875 BIOSIS
DOCUMENT NUMBER: PREV198937027647; BR37:27647
TITLE: INTERFERON-GAMMA STIMULATED PROTEIN PHOSPHORYLATION IN U937 CELLS DIFFERS FROM THAT STIMULATED BY IONOMYCIN OR PHORBOL DIESTERS.
AUTHOR(S): SCHEPERS T M [Reprint author]; FELDHOFF P A; DEAN W L; KLEIN J B; MCLEISH K R
CORPORATE SOURCE: DEP MED, UNIV LOUISVILLE SCH MED, LOUISVILLE, KY 40292, USA
SOURCE: FASEB Journal, (1989) Vol. 3, No. 4, pp. A1290.
Meeting Info.: 73RD ANNUAL MEETING OF THE FEDERATION OF AMERICAN SOCIETIES FOR EXPERIMENTAL BIOLOGY, NEW ORLEANS, LOUISIANA, USA, MARCH 19-23, 1989. FASEB (FED AM SOC EXP BIOL) J.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 11 Jul 1989
Last Updated on STN: 14 Jul 1989

L20 ANSWER 44 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1989:625466 HCAPLUS
DOCUMENT NUMBER: 111:225466
TITLE: Sn-1,2-diacylglycerols and phorbol ester stimulate the production of progesterone from the human placenta
AUTHOR(S): Kato, Hiromi; Kato, Masayuki; Kasugai, Masahide; Mizutani, Sigehiko; Ninagawa, Terumi; Tomoda, Yutaka
CORPORATE SOURCE: Sch. Med., Nagoya Univ., Nagoya, 466, Japan
SOURCE: Acta Endocrinologica (1989), 121(4), 560-6
CODEN: ACENA7; ISSN: 0001-5598
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Human term placental explants were used to investigate the possible role of phospholipid-sensitive and Ca2+-dependent protein kinase in the regulation of human placental progesterone production Placental tissue was incubated with low-d. lipoprotein as a precursor of progesterone in the presence or the absence of phorbol 12-myristate-13-acetate, 1-oleoyl-2-acetyl-glycerol, and the Ca ionophore A23187. The rate of progesterone production by placental tissue was 21.7 ng/mg wet wt/(2 h with 500 mg low d. lipoprotein/L (control). The rate of of progesterone production was accelerated 2-fold by 1 nmol/L phorbol 12-myristate-3-acetate, 1.6-fold by 250 μmol/L 1-oleoyl-2-acetyl-glycerol and this increase was dose-related (25-250 μmol/L 1-oleoyl-2-acetyl-glycerol). A nonpromoting derivative, 4α-phorbol 12,13-didecanoate had no effect. The phorbol 12-myristate-13-acetate or 1-oleoyl-2-acetyl-glycerol-induced stimulation of progesterone production was not associated with a change in the

intracellular cAMP level. Addition of 10 μmol/L A23187 further increased progesterone production with 125 μmol/L 1-oleoyl-2-acetyl-glycerol. The rate of progesterone production was accelerated 1.6-fold by 125 μmol/L 1-oleoyl-2-acetyl-glycerol and 10 μmol/L A 23187 as compared with control. The effects of the phorbol ester and the diacyl glycerol were completely blocked by the addition of the protein synthesis inhibitor cycloheximide. Apparently, these phorbol reagents are able to stimulate human placental progesterone production The possible roles of intracellular Ca2+ and protein kinase C in placental steroidogenesis are discussed.

L20 ANSWER 45 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1989:151824 HCAPLUS
DOCUMENT NUMBER: 110:151824
TITLE: Phosphorylation-dependent and -independent pathways of platelet aggregation
AUTHOR(S): Watson, Stephen P.; Hambleton, Sophie
CORPORATE SOURCE: Dep. Pharmacol., Univ. Oxford, Oxford, OX1 3QT, UK
SOURCE: Biochemical Journal (1989), 258(2), 479-85
CODEN: BIJOAK; ISSN: 0306-3275
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The nonspecific inhibitor of protein kinases, staurosporine, was used to investigate the role of protein phosphorylation during aggregation, the mobilization of intracellular Ca2+ (Ca2+i), and intracellular pH (pHi) in thrombin-stimulated human platelets. The concentration of staurosporine chosen for these studies, 1 μM, was previously reported to inhibit protein phosphorylation completely but to have no effect on the activation of phospholipase C in thrombin-stimulated human platelets. Aggregation induced by phorbol dibutyrate is slow (several minutes) and is inhibited completely by staurosporine. In contrast, aggregation induced by thrombin, platelet-activating factor, or ionophore A 23187 is rapid (occurs within 60 s), and is slowed, but not inhibited, in the presence of staurosporine. On the other hand, staurosporine causes a small potentiation of the peak [Ca2+]i signal induced by thrombin and a marked increase in the half-life of decay of this signal, but has no effect on pHi. Under conditions designed to prevent an increase in [Ca2+]i (presence of Ni2+ to prevent Ca2+ entry, and depletion of the intracellular Ca2+ stores), aggregation induced by thrombin resembles that by phorbol dibutyrate and is now inhibited completely by staurosporine. Evidently, there are 2 signalling pathways for aggregation, a relatively rapid phosphorylation-independent route mediated by Ca2+ and a slower, phosphorylation-dependent, pathway mediated by protein kinase C. Since staurosporine slows aggregation induced by thrombin, it appears that under normal conditions these pathways interact synergistically.

L20 ANSWER 46 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1989:527664 HCAPLUS
DOCUMENT NUMBER: 111:127664
TITLE: Protein kinase C activation alters the sensitivity of agonist-stimulated endothelial-cell prostacyclin production to intracellular calcium
AUTHOR(S): Carter, Thomas D.; Hallam, Trevor J.; Pearson, Jeremy D.
CORPORATE SOURCE: Sect. Vascular Biol., MRC Clin. Res. Cent., Harrow/Middlesex, HA1 3UJ, UK
SOURCE: Biochemical Journal (1989), 262(2), 431-7
CODEN: BIJOAK; ISSN: 0306-3275
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Agonist-stimulated release of prostacyclin (PGI2) from endothelial cells requires elevation of the concentration of intracellular ionized calcium ([Ca2+]i) above a threshold value, and raised [Ca2+]i provides a sufficient transduction signal to account for the extent of PGI2 production However, chronic activation of protein kinase C has been reported sep. to

potentiate PGI2 release, but to depress agonist-induced elevations of [Ca2+]i. Pretreatment with phorbol 12-myristate 13-acetate (PMA) dose-dependently induces PGI2 release over many minutes after a significant lag period without any change in [Ca2+]i. In addition, PMA potentiates the transient release of PGI2 in response to agonists in a complex manner depending on the time of preincubation and the concns. of both PMA and agonist. Concomitant measurement of [Ca2+]i and PGI2 release demonstrates that PMA pretreatment dose-dependently inhibits both the peak [Ca2+]i transient and the subsequent steady-state elevation of [Ca2+]i in response to agonists. Determination of the quant. [Ca2+]i/PGI2 dose/response relationship, when PGI2 release is driven purely by elevating [Ca2+]i with ionomycin, demonstrates that PMA also enhances the Ca2+ sensitivity of PGI2 release. The observed effects of PMA on PGI2 release can be explained quant. by its abilities to lower the threshold [Ca2+]i required for PGI2 synthesis and to depress the peak [Ca2+]i evoked by agonist. These effects are due resp. to actions of PMA on phospholipase A2 and on a G-protein (Gp) that couples activated receptors to phospholipase C.

L20 ANSWER 47 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1989:350028 BIOSIS
DOCUMENT NUMBER: PREV198937041125; BR37:41125
TITLE: SIGNALING VIA THE T-CELL ANTIGEN RECEPTOR HETERODIMER AND THE CD2 ANTIGEN A NOVEL SYNERGISTIC PATHWAY FOR ACTIVATION OF T-CELLS.
AUTHOR(S): SUTHANTHIRAN M [Reprint author]
CORPORATE SOURCE: ROGOSIN INST, CORNELL UNIV MED COLL, NEW YORK, NY 10021, USA
SOURCE: Transplantation Proceedings, (1989) Vol. 21, No. 1 PART 1, pp. 340-341.
Meeting Info.: TWELFTH INTERNATIONAL CONGRESS OF THE TRANSPLANTATION SOCIETY, SYDNEY, NEW SOUTH WALES, AUSTRALIA, AUGUST 14-19, 1988. TRANSPLANT PROC.
CODEN: TRPPA8. ISSN: 0041-1345.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 25 Jul 1989
Last Updated on STN: 29 Jul 1989

L20 ANSWER 48 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1989:476350 HCAPLUS
DOCUMENT NUMBER: 111:76350
TITLE: Transforming growth factors β1 and β2 as well as milk growth factor decrease anti-CD3-induced proliferation of human lymphocytes without inhibiting the anti-CD3-mediated increase of [Ca2+]i and the activation of protein kinase C
AUTHOR(S): Stoeck, Michael; Sommermeyer, Henning; Miescher, Sylvia; Cox, David; Alkan, Sefik; Szamel, Marta
CORPORATE SOURCE: Lausanne Branch, Ludwig Inst. Cancer Res., Epalinges, CH-1066, Switz.
SOURCE: FEBS Letters (1989), 249(2), 289-92
CODEN: FEBLAL; ISSN: 0014-5793
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Porcine transforming growth factors 1 and 2 (pTGF-β1 and β2) and milk growth factor (MGF) at 1 ng/mL significantly inhibited the proliferation of human lymphocytes induced by anti-CD3 antibodies. In contrast, the anti-CD3-mediated increase of intracellular Ca2+ and the activation and translocation of protein kinase C were not affected by the transforming growth factors.

L20 ANSWER 49 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1990:19364 HCAPLUS

DOCUMENT NUMBER: 112:19364
TITLE: Involvement of protein kinase C in translocation of desmoplakins from cytosol to plasma membrane during desmosome formation in human squamous cell carcinoma cells grown in low to normal calcium concentration
AUTHOR(S): Sheu, Hammming; Kitajima, Yasuo; Yaoita, Hideo
CORPORATE SOURCE: Dep. Dermatol., Jichi Med. Sch., Tochigi, Japan
SOURCE: Experimental Cell Research (1989), 185(1), 176-90
CODEN: ECREAL; ISSN: 0014-4827
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The intracellular signal transduction mechanism leading to desmosome formation in low calcium-grown keratinocytes after addition of calcium to the medium was studied by immunofluorescence using antibodies to desmoplakins I and II (cytoplasmic desmosomal proteins) and by electron microscopy before and after addition of: calcium; protein kinase C (PKC) activators 12-O-tetradecanoylphorbol-13-acetate (TPA), phorbol-12,13-dibutyrate (PDBu), and 1,2-dictanoylglycerol (DOG); calcium ionophore A23187; selective PKC inhibitors 1-(5-isoquinolinylsulfonyl)-2-methylpiperazine (H-7) and staurosporine; and a Ca2+/calmodulin-dependent kinase inhibitor, N-(6-aminohexyl)-5-chloro-1-naphthalenesulfonamide (W-7). In previous studies using a low-calcium-grown human epidermal squamous cell carcinoma, it was shown that an increase in extracellular Ca2+ caused a four-fold increase in PKC activity and addition of TPA (10 ng/mL) induced a transient increase in membrane-bound PKC activity in association with cell-cell contact formation. The present study showed that TPA (10 ng/mL), PDBu (10 ng/mL), and DOG (1 mg/mL) induced a rapid cell-cell contact and redistribution of desmoplakins from cytoplasm to the plasma membrane with desmosome formation within 60-120 min, which was similar, although less marked, to the effect of increased Ca2+. The TPA-induced desmosome formation was inhibited by selective PKC inhibitors, H-7 (20 μM) or staurosporine (100 nM). On the other hand, calcium ionophore A23187 induced only a temporary increase in the number of desmoplakin-containing fluorescent spots in the cytoplasm and a temporary cell-cell attachment without desmosome formation. The calcium-induced desmosome formation was partially inhibited by 20-100 μM H-7 or 100 nM staurosporine; however, it was not inhibited by W-7 at a concentration of 25 μM, at which this agent selectively inhibits calmodulin-dependent protein kinase. These results suggest that PKC activation plays an important role in desmoplakin translocation from the cytoplasm to the plasma membrane as one of the processes of calcium-induced desmosome formation.

L20 ANSWER 50 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1989:587749 HCAPLUS
DOCUMENT NUMBER: 111:187749
TITLE: Effect of phorbol ester TPA and calcium ionophore A 23187 on the progesterone production of granulosa luteal cells in vitro
AUTHOR(S): Zimmermann, Gerolf; Alexander, Henry; Weber, Wolfgang; Schoene, Elfrun; Giese, Thomas; Haake, Karl-Wilhelm
CORPORATE SOURCE: Bereich Med., Karl-Marx-Univ., Leipzig, DDR-7010, Ger. Dem. Rep.
SOURCE: Wissenschaftliche Zeitschrift - Karl-Marx-Universitaet Leipzig, Mathematisch-Naturwissenschaftliche Reihe (1989), 38(2), 143-52
CODEN: WZMNA8; ISSN: 0043-6860
DOCUMENT TYPE: Journal
LANGUAGE: German

AB For some animal species it could be confirmed that with the LH surge and beginning progesterone (PROG) synthesis along with the adenylate cyclase/cAMP system, there is another second messenger system of protein kinase C/Ca2+. Both systems regulate simultaneously the PROG synthesis of the different developmental states of luteal cells. Results of cell cultivation show that human granulosa luteal cells following in vitro

fertilization (IVF) stimulation respond to both regulation systems in vitro. Human chorionic gonadotropin (HCG) (10 IU/mL) and exogenous dibutyryl-cAMP (4 mmol/L) added in the initial phases of culture increase the PROG production of the cells, though with a lower induction effect to the falling fertilization rates of the IVF cycles. The analogs of protein kinase C action, TPA (40 nmol/L) and Ca ionophore A 23187 (400 nmol/L) form, primarily only in the subsequent incubation days, lower PROG levels in the culture medium. The inhibiting effect is markedly pronounced when pregnant groups were compared to those without fertilization. The in-vitro findings demonstrate that the minimally stimulated IVF cycles are able to increase the HCG-induced PROG synthesis readily without the activation of the PROG-inhibiting protein kinase C-system. Therefore, HCG application as a support of the luteal phase of IVF cycles is generally available for optimally stimulated patients. On the other hand, unfavorably high LH and PROG concns. following human menopausal gonadotropin stimulation can result in a further inhibition of PROG secretion and diminution of the luteal phase.

L20 ANSWER 51 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1988:588600 HCAPLUS
DOCUMENT NUMBER: 109:188600
TITLE: Coordination and reversibility of signals for proliferative activation and interleukin 2 mRNA production in resting human T lymphocytes by phorbol ester and calcium ionophore
AUTHOR(S): McCrady, Carl W.; Ely, Constance M.; Westin, Eric; Carchman, Richard A.
CORPORATE SOURCE: Med. Coll. Virginia, Virginia Commonw. Univ., Richmond, VA, 23298, USA
SOURCE: Journal of Biological Chemistry (1988), 263(34), 18537-44
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Sequential stimulation and washout procedures were employed to examine the kinetics and reversibility of pharmacol. manipulated 2nd messenger signals mediating phenotypic changes and proliferative activation of resting human T lymphocytes. Phorbol dibutyrate (PDBu) was used to stimulate protein kinase C (Ca2+/phospholipid-dependent enzyme) while ionomycin was used to manipulate intracellular Ca2+ levels. Stimulation by PDBu alone induced phosphorylation of several endogenous substrates and altered expression of phenotypic markers, downregulating expression of CD4 and CD3 while increasing expression of CD2 and the interleukin 2 (IL-2) receptor. Stimulation with ionomycin alone caused an increase in intracellular Ca2+ levels but did not induce proliferation or cause major changes in the expression of phenotypic markers. Anal. of endogenous PDBu stimulated phosphosubstrates indicated that some substrates (pp92, pp82, pp55) underwent dephosphorylation, returning to base-line levels following PDBu removal while others (pp61, pp65) showed only partial dephosphorylation, and one (pp28) remained phosphorylated. Washing ionomycin-stimulated cells resulted in an approx. 75% reduction of intracellular Ca2+. Ionomycin exposure did not alter the affinity or number of receptors for [3H]PDBu. These data suggest that signals induced by PDBu or ionomycin are reversible following removal of the stimulating agents with respect to proliferative activation of T lymphocytes. A pretranscription mechanism regulating the production of IL-2 mRNA requires simultaneous activation of protein kinase C and elevation of intracellular Ca2+.

L20 ANSWER 52 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1989:28953 BIOSIS
DOCUMENT NUMBER: PREV198987016953; BA87:16953
TITLE: COMPARISON OF SIGNALS DELIVERED THROUGH CD3 AND CD2 FOR T-CELL ACTIVATION THE ROLE OF CALCIUM INFLUX AND INTERLEUKIN 1.

AUTHOR(S): WAKASUGI H [Reprint author]; MAHE Y; HUET S; BOUMSELL L; BERNARD A; TURSZ T
CORPORATE SOURCE: LABORATOIRE D'IMMUNOBIOLOGIE DES TUMEURS, UA 1156 CNRS, INST GUSTAVE-ROUSSY, RUE CAMILLE DESMOULINS 94805 VILLEJUIF CEDEX, FRANCE
SOURCE: Human Immunology, (1988) Vol. 23, No. 3, pp. 163-178. CODEN: HUIMDQ. ISSN: 0198-8859.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 20 Dec 1988
Last Updated on STN: 20 Dec 1988

AB In the absence of monocytes, resting T lymphocytes extensively purified from human peripheral blood failed to proliferate when stimulated with a mixture of calcium ionophore, which elevates intracellular calcium levels, and TPA, which activated protein kinase C. A third signal, i.e., the triggering via CD3 or CD2 molecules, was necessary in order to observe proliferation. These highly purified T cells required the presence of monocytes in both CD3 and CD2 systems for their proliferation. Exogenous interleukin 1 clearly substituted for monocytes in CD1- but not in CD3-triggered T-cell proliferation. In contrast, the effect of CD2 and CD3 antibodies on Ca-+ influx was apparently not dependent on the presence of monocytes. In the presence or absence of the monocytes, CD3, as well as certain combinations of CD2 monoclonal antibodies including the D66 monoclonal antibody, were able to increase the intracellular calcium concentration as measured by Quin 2 fluorescence. EGTA, a Ca++ chelator, completely inhibited CD2- and CD3-mediated T-cell proliferation, indicating that calcium uptake is necessary during the T-cell proliferation. The addition of TPA abrogated the inhibitory effect of EGTA and completely restored the response of the T cells stimulated by CD3, but not by CD2, monoclonal antibodies. In the CD2 pathway, EGTA-inhibited proliferation of T cells could be completely restored by addition of exogenous interleukin 2 as well as exogenous recombinant interleukin 1. Our results indicate that EGTA inhibits the production of interleukin 1 but has no direct effect on either interleukin 2 production or on Tac antigen expression. In this system, recombinant interleukin 1 alpha demonstrated a more potent ability for restoring the T-cell response than did recombinant interleukin 1 beta. These results suggest that interleukin 1 could act as a potent costimulatory factor in the non-antigen-specific T-cell activation.

L20 ANSWER 53 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1988:605326 HCAPLUS
DOCUMENT NUMBER: 109:205326
TITLE: Calcitonin gene-related peptide and calcitonin secretion from a human medullary thyroid carcinoma cell line: effects of ionomycin, phorbol ester and forskolin
AUTHOR(S): Haller-Brem, S.; Muff, R.; Fischer, J. A.
CORPORATE SOURCE: Res. Lab. Calcium Metab., Dep. Orthopedic Surg., Zurich, 8008, Switz.
SOURCE: Journal of Endocrinology (1988), 119(1), 147-52 CODEN: JOENAK; ISSN: 0022-0795
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In a human medullary thyroid carcinoma (MTC) cell line, ionomycin (10 μmol/L) raised the cytosolic free Ca concentration (Cai2+) concomitant with a transient stimulation of the secretion of calcitonin gene-related peptide (CGRP) and calcitonin whereas TPA (16 nmol/L) did not affect the concentration of Cai2+ but caused a gradual rise in the secretion of CGRP and calcitonin. Combined addition of 10 μmol ionomycin/L and 16 nmol TPA/L resulted in additive stimulation of CGRP and calcitonin secretory responses. Forskolin (10 μmol/L) alone did not change the concentration of Cai2+,

marginally enhanced the release of CGRP and calcitonin, and increased by 23-fold the cellular levels of cAMP. Ionomycin and TPA did not change cellular cAMP. Forskolin synergistically enhanced the ionomycin-induced early phase as well as the TPA-induced late phase of the CGRP and calcitonin secretory responses. Thus, increased concns. of Cai2+ together with protein kinase C and A activation mediate the secretion of CGRP and calcitonin in MTC cells.

L20 ANSWER 54 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1987:182708 BIOSIS
DOCUMENT NUMBER: PREV198732089835; BR32:89835
TITLE: SELECTIVE ACTIVITY OF PLANT FLAVONOIDS ON NEUTROPHIL CHEMILUMINESCENCE CL.
AUTHOR(S): CUMELLA J C [Reprint author]; FADEN H; MIDDLETON E JR
CORPORATE SOURCE: BUFFALO, NY, USA
SOURCE: Journal of Allergy and Clinical Immunology, (1987) Vol. 79, No. 1, pp. 157.
Meeting Info.: FORTY-THIRD ANNUAL MEETING OF THE AMERICAN ACADEMY OF ALLERGY AND IMMUNOLOGY, WASHINGTON, D.C., USA, FEB. 19-25, 1987. J ALLERGY CLIN IMMUNOL.
CODEN: JACIBY. ISSN: 0091-6749.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 14 Apr 1987
Last Updated on STN: 14 Apr 1987

L20 ANSWER 55 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1987:82947 HCAPLUS
DOCUMENT NUMBER: 106:82947
TITLE: Comparison of the roles of calmodulin and protein kinase C in activation of the human neutrophil respiratory burst
AUTHOR(S): Wright, Clifford D.; Hoffman, Michael D.
CORPORATE SOURCE: Pharmacol. Dep., Warner-Lambert/Parke-Davis Pharm. Res., Ann Arbor, MI, 48105, USA
SOURCE: Biochemical and Biophysical Research Communications (1987), 142(1), 53-62
CODEN: BBRCA9; ISSN: 0006-291X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The roles of calmodulin and protein kinase C in the activation of the human neutrophil respiratory burst were characterized pharmacol. The protein kinase C inhibitors 1-(5-isoquinolinylsulfonyl)-2-methylpiperazine (H-7) and N-(2-aminoethyl)-5-isoquinolinesulfonamide (H-9) did not inhibit superoxide anion generation by neutrophils stimulated for 30 min with N-formyl-L-Met-L-Leu-L-Phe (FMLP) or 4-β-phorbol 12-β-myristate 13-α-acetate (PMA). However, H-7 did depress superoxide production during the first 5 min following stimulation. In contrast, the specific calmodulin antagonist N-(6-aminohexyl)-5-chloro-1-naphthalenesulfonamide (W-7) and the dual calmodulin antagonist/protein kinase C inhibitor trifluoperazine (TFP) were potent inhibitors of the response throughout the 30 min incubation. Stimulation of neutrophils with submaximal doses of FMLP or PMA failed to promote inhibition of the respiratory burst by H-7 or H-9, but did stimulate a respiratory response which was not inhibited by TFP or W-7. Apparently, while protein kinase C may play a role in the initiation of the respiratory burst response, propagation of the response is dependent on the calmodulin-dependent processes. The inability of TFP and W-7 to inhibit superoxide anion generation in response to submaximal stimulatory doses of FMLP or PMA suggests that calmodulin-independent processes may also be involved in activation of the respiratory burst.

L20 ANSWER 56 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1986:531964 HCAPLUS
DOCUMENT NUMBER: 105:131964
TITLE: Activation and inhibition of 5-lipoxygenase in human polymorphs: comparison with protein kinase C
AUTHOR(S): Randall, Roger W.; Tateson, James E.; Garland, Lawrence G.
CORPORATE SOURCE: Dep. Biochem., Wellcome Res. Lab., Beckenham/Kent, BR3 3BS, UK
SOURCE: Biochemical Society Transactions (1986), 14(6), 1153-4
CODEN: BCSTB5; ISSN: 0300-5127
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The 5-lipoxygenase activity in intact polymorphs purified from human blood was biphasically affected by phorbol myristate acetate (PMA). At low doses (0.001-1 mM), PMA caused a dose-dependent stimulation of the enzyme, whereas at 10 and 100 mM, PMA inhibited the enzyme. The effects of PMA were dependent on the addition of the Ca ionophore A23187. Retinal also inhibited the enzyme. The effects of PMA and retinal did not appear to be via direct enzyme inhibition. The previously observed effects of PMA and retinal on protein kinase C are discussed with regard to a possible role of protein kinase C and Ca in regulating polymorph 5-lipoxygenase activity.

L20 ANSWER 57 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1986:265535 BIOSIS
DOCUMENT NUMBER: PREV198631010455; BR31:10455
TITLE: THE CALCIUM MESSENGER SYSTEM 1.
AUTHOR(S): RASMUSSEN H [Reprint author]
CORPORATE SOURCE: INTERNAL MED CELL BIOL, YALE UNIV SCH MED, 333 CEDAR ST, NEW HAVEN, CONN 06510, USA
SOURCE: New England Journal of Medicine, (1986) Vol. 314, No. 17, pp. 1094-1101.
CODEN: NEJMAG. ISSN: 0028-4793.
DOCUMENT TYPE: Article
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 28 Jun 1986
Last Updated on STN: 28 Jun 1986

L20 ANSWER 58 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1986:550375 HCAPLUS
DOCUMENT NUMBER: 105:150375
TITLE: Role of intracellular calcium and protein kinase C in the endocytosis of transferrin and insulin by HL60 cells
AUTHOR(S): Iacopetta, Barry; Carpentier, Jean Louis; Pozzan, Tullio; Lew, Daniel P.; Gorden, Phillip; Orci, Lelio
CORPORATE SOURCE: Inst. Histol. Embryol., Univ. Med. Cent., Geneva, 1211/4, Switz.
SOURCE: Journal of Cell Biology (1986), 103(3), 851-6
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The role of the cytosolic free Ca2+ concentration ([Ca2+]i) and of protein kinase C on the internalization of transferrin and insulin in the human promyelocytic cell line HL60 was investigated. [Ca2+]i was selectively monitored and manipulated by the use of the fluorescent Ca2+ indicator and buffer quin2, whereas receptor-ligand internalization was studied directly by quant. electron microscope autoradiog. Decreasing the [Ca2+]i ≤10-fold below the resting level had no effect on the internalization of transferrin or insulin. Similarly, a 10-fold elevation of the [Ca2+]i by using the Ca2+ ionophore ionomycin caused little or no change in the endocytosis f the 2 ligands. In contrast, activation of

protein kinase C by phorbol myristate acetate markedly stimulated the internalization of both occupied and unoccupied transferrin receptors, even in cells with very low [Ca2+]i. The insulin receptor behaved differently in response to phorbol myristate acetate, however, in that only the occupied receptors were stimulated to internalize. Thus, [Ca2+]i plays only a minor role in regulating receptor-mediated endocytosis, whereas protein kinase C can selectively modulate receptor internalization depending on receptor type and occupancy.

L20 ANSWER 59 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1986:184753 HCAPLUS
DOCUMENT NUMBER: 104:184753
TITLE: The protein kinase C inhibitors H-7 and H-9 fail to inhibit human neutrophil activation
AUTHOR(S): Wright, Clifford D.; Hoffman, Michael D.
CORPORATE SOURCE: Parke-Davis Pharm. Res. Div., Warner-Lambert Co., Ann Arbor, MI, 48105, USA
SOURCE: Biochemical and Biophysical Research Communications (1986), 135(3), 749-55
CODEN: BBRCA9; ISSN: 0006-291X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The protein kinase C inhibitors H 7 and H 9 were examined for their ability to inhibit human neutrophil activation. At concns. ≤100 mM, these compds. failed to inhibit either respiratory burst or the secretory response of neutrophils stimulated with particulate (serum-opsonized zymosan) or soluble (A23187, N-formyl-Met-Leu-Phe, PMA) stimuli. In contrast, the calmodulin antagonist W 7 inhibited both O2- generation and lysosomal enzyme release in response to the same stimuli. Thus, calmodulin-dependent enzymes, rather than protein kinase C, may be essential for neutrophil activation.

L20 ANSWER 60 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1986:236513 BIOSIS
DOCUMENT NUMBER: PREV198682001017; BA82:1017
TITLE: SPECIFIC CLEAVAGE OF THE FIBROBLAST RECEPTOR FOR PLATELET-DERIVED GROWTH FACTOR BY AN ENDOGENOUS CALCIUM-DEPENDENT THIOL PROTEASE.
AUTHOR(S): EK B [Reprint author]; HELDIN C-H
CORPORATE SOURCE: INSTITUTIONEN FOR MEDICINSK OCH FYSIOL KEMI, UPPSALA UNIV, BIOMEDICINSKA CENTRUM, BOX 575, S-751 23 UPPSALA, SWEDEN
SOURCE: European Journal of Biochemistry, (1986) Vol. 155, No. 2, pp. 409-414.
CODEN: EJBCAI. ISSN: 0014-2956.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 7 Jun 1986
Last Updated on STN: 7 Jun 1986
AB Previous studies have shown that platelet-derived growth factor (PDGF) stimulates the phosphorylation of two components in membranes prepared from human fibroblasts in the presence of Ca2+. One of these represents the 185-kDa PDGF receptor, which undergoes autophosphorylation, and the other has an Mr of 130000. We show in this communication that the 130-kDa component is derived from the 185-kDa receptor via proteolysis by an endogenous Ca2+-dependent protease, which is dependent on a reduced -SH group for its activity. The 130-kDa fragment contains several of the characteristics of the receptor, such as the PDGF-binding site and the major autophosphorylation sites. Furthermore, the cleaved receptor retains tyrosine kinase activity.

L20 ANSWER 61 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1986:201321 BIOSIS
DOCUMENT NUMBER: PREV198681092621; BA81:92621

TITLE: LYMPHOCYTE ACTIVATION BY OKT-3 CYCLOSPORINE SENSITIVITY AND SYNERGISM WITH PHORBOL ESTER.
AUTHOR(S): KAY J E [Reprint author]; BENZIE C R
CORPORATE SOURCE: SCH BIOLOGICAL SCI, UNIV SUSSEX, BRIGHTON, BN1 9QG, UK
SOURCE: Immunology, (1986) Vol. 57, No. 2, pp. 195-200.
CODEN: IMMUAM. ISSN: 0019-2805.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 28 May 1986
Last Updated on STN: 28 May 1986

AB Lymphocyte activation by the mitogenic monoclonal antibody OKT3 is less effective than activation by mitogenic lectins such as phytohaemagglutinin (PHA) and concanavalin A (Con A). Activation by OKT3 is also very sensitive to inhibition by cyclosporin (CSA), which selectively inhibits Ca2+-activated steps in the activation process. In addition, the magnitude of the OKT3 response can be raised to that seen with mitogenic lectins by coincubation with phorbol esters (which activate protein kinase C). These observations suggest that OKT3 may deliver efficiently that Ca2+ signal involved in the initiation of lymphocyte activation, and that the comparatively weak overall response is due to a failure to generate a second signal, probably the activation of protein kinase C, as efficiently as the mitogenic lectins.

L20 ANSWER 62 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1986:49707 HCAPLUS
DOCUMENT NUMBER: 104:49707
TITLE: The role of calcium and calcium-activated phospholipid-dependent protein kinase in degranulation of human neutrophils
AUTHOR(S): Kang, Dongchon; Tsuda, Hiroko; Takeshige, Koichiro; Shibata, Yosaburo; Minakami, Shigeki
CORPORATE SOURCE: Sch. Med., Kyushu Univ., Fukuoka, 812, Japan
SOURCE: Journal of Biochemistry (Tokyo, Japan) (1985), 98(6), 1699-706
CODEN: JOBIAO; ISSN: 0021-924X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The degranulation reactions of human neutrophils induced by 1-oleoyl-2-acetyl-glycerol (OAG), phorbol 12-myristate 13-acetate (PMA), and Ca2+ ionophore A23187 or their combinations were studied. OAG in the absence of A23187 stimulated the release of both lysozyme and lactoferrin, constituents of the specific granules, but it did not stimulate the release of β-glucuronidase, an enzyme of the azurophil granules. Electron microscopy revealed a selective decrease in the nos. of the specific granules in this case. The combined effects of A23187 at a concentration >0.1 μM and OAG were essentially additive. W-7, known to be an inhibitor of both Ca2+-activated phospholipid-dependent protein kinase (C-kinase) and calmodulin, inhibited the degranulation induced by OAG or PMA, but it inhibited the reaction induced by A23187 less markedly. The release of lysozyme reached a plateau at about 0.1 μM A23187 and increased again at higher concns. of A23187. Thus, degranulation can be induced by the activation of the C-kinase, and the degranulation by A23187 at low concns. may be due to the activation of the C-kinase; the effects of A23187 at high concns., however, could not be explained only in terms of the activation of the C-kinase.

L20 ANSWER 63 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1985:517005 HCAPLUS
DOCUMENT NUMBER: 103:117005
TITLE: Synergistic stimulation of thromboxane biosynthesis by calcium ionophore and phorbol ester or thrombin in human platelets
AUTHOR(S): Mobley, Alice; Tai, Hsin Hsiung

CORPORATE SOURCE: Coll. Pharm., Univ. Kentucky, Lexington, KY, 40536-0053, USA
SOURCE: Biochemical and Biophysical Research Communications (1985), 130(2), 717-23
CODEN: BBRCA9; ISSN: 0006-291X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB PMA and low concns. of the Ca2+ ionophore A 23187, which given sep. have minimal effect in stimulating thromboxane synthesis in human platelets, showed marked synergism when given simultaneously. A similar synergism was also demonstrated between thrombin [9002-04-4] or collagen and low concns. of A 23187, but not of PMA. Simultaneous addition of thrombin and PMA results in less synthesis of thromboxane than addition of thrombin alone. Apparently, protein kinase [9026-43-1] C activation by agonists may not only induce, but also regulate thromboxane synthesis in human platelets.

L20 ANSWER 64 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1985:447950 HCAPLUS
DOCUMENT NUMBER: 103:47950
TITLE: Superoxide generation by either 1-oleoyl-2-acetylglycerol or A23187 in human neutrophils is enhanced by indomethacin
AUTHOR(S): Dale, M. Maureen; Penfield, Adrienne
CORPORATE SOURCE: Dep. Pharmacol., Univ. Coll. London, London, WC1E 6BT, UK
SOURCE: FEBS Letters (1985), 185(1), 213-17
CODEN: FEBLAL; ISSN: 0014-5793
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Indomethacin [53-86-1], at a concentration (10-4M) which depressed the O2-generation induced by fMet-Leu-Phe in human neutrophils, markedly enhanced the O-2 generation induced by both 1-oleoyl-2-acetylglycerol and the Ca2+ ionophore A23187. These results are explicable in terms of the hypothesis that synergism between cytosolic Ca2+ and protein kinase [9026-43-1] C is involved in signal transduction for the respiratory burst in neutrophils.

L20 ANSWER 65 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1984:548854 HCAPLUS
DOCUMENT NUMBER: 101:148854
TITLE: Dependence of exocytosis on cytosolic calcium and protein kinase c activation in human neutrophils
AUTHOR(S): Di Virgilio, Francesco; Lew, P. Daniel; Pozzan, Tullio
CORPORATE SOURCE: Unit Study Physiol. Mitochondria, CNR, Padua, Italy
SOURCE: Biochemical Society Transactions (1984), 12(6), 1078
CODEN: BCSTB5; ISSN: 0300-5127
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The releasing activity of fMet-Leu-Phe on the secondary granule content (vitamin B12-binding protein) in human neutrophils was almost completely abolished by lowering the cytosolic free Ca2+ concentration to 10-8M, whereas the response to phorbol myristate acetate was fully conserved. Since phorbol myristate acetate elicited a full metabolic response even at negligible cytosolic free Ca2+ concns., the phorbol ester apparently activates protein kinase c by itself and does not only increase its affinity to Ca2+.

L20 ANSWER 66 OF 68 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1984:608928 HCAPLUS
DOCUMENT NUMBER: 101:208928
TITLE: Synergism between phorbol ester and A23187 in superoxide production by neutrophils
AUTHOR(S): Dale, M. Maureen; Penfield, Adrienne
CORPORATE SOURCE: Dep. Pharmacol., Univ. Coll. London, London, WC1E 6BT,

UK
SOURCE: FEBS Letters (1984), 175(1), 170-2
CODEN: FEBLAL; ISSN: 0014-5793
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Concns. of phorbol myristate acetate and the calcium ionophore, A23187, which by themselves are minimally effective in stimulating superoxide generation in human neutrophils show marked mutual potentiation when given together. This supports the hypothesis that synergism between cytosolic calcium and protein kinase C is involved in the stimulus/activation coupling of the respiratory burst in the neutrophil.

L20 ANSWER 67 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1984:83924 BIOSIS
DOCUMENT NUMBER: PREV198427000416; BR27:416
TITLE: PLATELET PROTEIN PHOSPHORYLATION AND CYTOPLASMIC CALCIUM.
AUTHOR(S): DANIEL J L [Reprint author]; SALGANICOFF L
CORPORATE SOURCE: THROMBOSIS CENTER, TEMPLE UNIVERSITY, PHILADELPHIA, PA USA, USA
SOURCE: Thrombosis and Haemostasis, (1983) Vol. 50, No. 1, pp. 92. Meeting Info.: 9TH INTERNATIONAL CONGRESS ON THROMBOSIS AND HEMOSTASIS, JULY 4-8, 1983. THROMB HEMOSTASIS.
CODEN: THHADQ. ISSN: 0340-6245.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH

L20 ANSWER 68 OF 68 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1985:149733 BIOSIS
DOCUMENT NUMBER: PREV198529039729; BR29:39729
TITLE: INTERACTION OF CYCLIC AMP AND CALCIUM-CALMODULIN IN THE ACTION OF VASOPRESSIN.
AUTHOR(S): SCHLONDORFF D [Reprint author]; LEVINE S L; SALISBURY J
CORPORATE SOURCE: ALBERT EINSTEIN COLL MED, NEW YORK, NY, USA
SOURCE: Adv. Nephrol. Necker Hosp., pp. 319-340. BACH, J. ET AL. (ED.). ADVANCES IN NEPHROLOGY: FROM THE NECKER HOSPITAL, VOL. 13. XXIII+383P. YEAR BOOK MEDICAL PUBLISHERS, INC.: CHICAGO, ILL., USA. ILLUS. 1984 (RECD. 1985).
Publisher: Series: Advances in Nephrology from the Necker Hospital.
CODEN: ANGYBQ. ISSN: 0084-5957. ISBN: 0-8151-4136-X.
DOCUMENT TYPE: Book
FILE SEGMENT: BR
LANGUAGE: ENGLISH

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(FILE 'HOME' ENTERED AT 08:12:09 ON 04 MAY 2004)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 08:12:39 ON 04 MAY 2004

L1	2087394	S	CALCIUM OR CALMODULIN
L2	38966	S	L1 (2W) KINASE?
L3	8783	S	HUMAN AND L2
L4	6504693	S	CLON? OR EXPRESS? OR RECOMBINANT
L5	4552	S	L3 AND L4
L6	4062032	S	BRAIN OR LUNG OR HIPPCAMPUS
L7	771	S	L5 AND L6
L8	78	S	HUMAN(W)L2
L9	17	S	L7 AND L8
L10	8		DUP REM L9 (9 DUPLICATES REMOVED)
		E	YAN C/AU
L11	996	S	E3

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                E KETCHUM K/AU
L12          193 S E7-E9
                E MERKULOV G/AU
L13           79 S E7-E9
                E BEASLEY E M/AU
L14          291 S E3
L15         1449 S L11 OR L12 OR L13 OR L14
L16            5 S L3 AND L15
L17            4 DUP REM L16 (1 DUPLICATE REMOVED)
                SET NOTICE DISPLAY 1

     INDEX 'IFICLS, PATOSEP, PATDPA, INPADOC' ENTERED AT 08:25:38 ON 04 MAY
     2004
                SEA US 6387677/PN,APPS
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               1   FILE IFICLS
               1   FILE INPADOC
L18             QUE US 6387677/PN,APPS
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L19            2 S L18
                SET NOTICE LOGIN DISPLAY

     FILE 'MEDLINE, EMBASE, BIOSIS, SCISEARCH, HCAPLUS, LIFESCI' ENTERED AT
     08:28:57 ON 04 MAY 2004
L20           68 DUP REM L8 (10 DUPLICATES REMOVED)
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	Issue Date	Pages	Document ID	Title
1	20040429	48	US 20040081999 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
2	20040429	84	US 20040081983 A1	Kinases and phosphatases
3	20040422	151	US 20040077044 A1	Kinases and phosphatases
4	20040415	337	US 20040072160 A1	Molecular toxicology modeling
5	20040408	53	US 20040067568 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
6	20040408	47	US 20040067522 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
7	20040401	68	US 20040063142 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
8	20040401	53	US 20040063130 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
9	20040325	81	US 20040058426 A1	Human kinases

	Issue Date	Pages	Document ID	Title
10	20040318	144	US 20040053394 A1	Human kinases
11	20040318	617	US 20040052820 A1	Fusion proteins comprising DP-178 and other viral fusion inhibitor peptides useful for treating aids
12	20040304	184	US 20040043466 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
13	20040226	152	US 20040038881 A1	Human kinases
14	20040226	52	US 20040038363 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
15	20040226	40	US 20040038362 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
16	20040226	259	US 20040038207 A1	Gene expression in bladder tumors
17	20040219	234	US 20040034196 A1	98 human secreted proteins
18	20040219	889	US 20040033235 A1	Nucleic acids encoding DP-178 and other viral fusion inhibitor peptides useful for treating aids
19	20040212	81	US 20040029280 A1	Viral vectors with modified tropism

	Issue Date	Pages	Document ID	Title
20	20040212	570	US 20040029114 A1	Methods of diagnosis of breast cancer, compositions and methods of screening for modulators of breast cancer
21	20040205	144	US 20040023242 A1	Human kinases
22	20040129	112	US 20040018185 A1	Human kinases
23	20040122		US 20040014659 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
24	20040122		US 20040014193 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
25	20040115	73	US 20040010136 A1	Composition for the detection of signaling pathway gene expression
26	20040115	484	US 20040009479 A1	Methods and compositions for diagnosing or monitoring auto immune and chronic inflammatory diseases
27	20040115		US 20040009477 A1	Methods for producing libraries of expressible gene sequences
28	20031218	111	US 20030232408 A1	ISOLATED HUMAN KINASE PROTEINS

	Issue Date	Pages	Document ID	Title
29	20031218		US 20030232336 A1	Novel human ion channel and transporter family members
30	20031211		US 20030228674 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
31	20031211	122	US 20030228595 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
32	20031113	136	US 20030211093 A1	Human kinases
33	20031113		US 20030211040 A1	Phosphodiesterase activity and regulation of phosphodiesterase 1B-mediated signaling in brain
34	20031106	128	US 20030207311 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
35	20031106	148	US 20030207299 A1	Human kinases

	Issue Date	Pages	Document ID	Title
36	20030925		US 20030180786 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
37	20030918		US 20030175927 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
38	20030918		US 20030175926 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
39	20030918		US 20030175791 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
40	20030904		US 20030166540 A1	Polynucleotide encoding a novel human G-protein coupled receptor, HGPRBMY30
41	20030904		US 20030166222 A1	39267, human kinase family members and uses therefor
42	20030904		US 20030166221 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
43	20030904		US 20030166219 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
44	20030904		US 20030166218 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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45	20030904	85	US 20030166215 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
46	20030821		US 20030157679 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
47	20030731		US 20030143690 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
48	20030731		US 20030143217 A1	Compositions containing nucleic acids and ligands for therapeutic treatment
49	20030724		US 20030140354 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
50	20030717		US 20030134784 A1	Nucleic acids and corresponding proteins entitled 83P2H3 and CaTrF2E11 useful in treatment and detection of cancer
51	20030717		US 20030134319 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
52	20030717		US 20030134302 A1	Libraries of expressible gene sequences
53	20030710		US 20030129704 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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54	20030710	90	US 20030129645 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
55	20030626		US 20030119037 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
56	20030508		US 20030087294 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
57	20030424		US 20030077799 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
58	20030417		US 20030073163 A1	Libraries of expressible gene sequences
59	20030403		US 20030064946 A1	Method and reagent for the inhibition of calcium activated chloride channel-1 (CLCA-1)
60	20030403	68	US 20030064475 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
61	20030327		US 20030059861 A1	Nucleotide sequences encoding mammalian calcium activated chloride channel-adhesion molecules
62	20030320		US 20030054529 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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63	20030313		US 20030049795 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
64	20030313		US 20030049792 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
65	20030306		US 20030044911 A1	Calcium channel compositions and methods of use thereof
66	20030227		US 20030040496 A1	Methods for halting unwanted cell growth, such as using ligand-directed nucleic acid delivery vehicles
67	20030227		US 20030040089 A1	Protein-protein interactions in adipocyte cells
68	20030206		US 20030027307 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
69	20030130		US 20030022341 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
70	20030130		US 20030022340 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
71	20030130		US 20030022339 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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72	20030130		US 20030022337 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
73	20030130		US 20030022232 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
74	20030130		US 20030022229 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
75	20030102		US 20030003560 A1	Isolated human casein kinase proteins, nucleic acid molecules encoding human casein kinase proteins, and uses thereof
76	20021219		US 20020192749 A1	Human polynucleotides, polypeptides, and antibodies
77	20021114		US 20020168741 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
78	20021024		US 20020155539 A1	Calcium channel polynucleotides, polypeptides, and antibodies
79	20021017		US 20020151020 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
80	20021003		US 20020142430 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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81	20021003		US 20020142427 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
82	20020926		US 20020137167 A1	ISOLATED HUMAN CASEIN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN CASEIN KINASE PROTEINS, AND USES THEREOF
83	20020919		US 20020132325 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
84	20020919		US 20020132324 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
85	20020919		US 20020132322 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
86	20020912		US 20020127683 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
87	20020905		US 20020123121 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
88	20020905		US 20020123120 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
89	20020829		US 20020119548 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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90	20020829		US 20020119544 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
91	20020815		US 20020110889 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
92	20020815		US 20020110888 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
93	20020801		US 20020103116 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
94	20020718		US 20020094946 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
95	20020718		US 20020094560 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
96	20020704		US 20020086391 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEROF
97	20020627		US 20020082189 A1	ISOLATED HUMAN SERINE/THREONINE KINASE NUCLEIC ACID MOLECULES ENCODING HUMAN SERINE/THREONINE KINASE AND USES THEREOF

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98	20020620		US 20020076783 A1	Plants and plants cells expressing histidine tagged intimin
99	20020613	68	US 20020072491 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
100	20020530		US 20020064851 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
101	20020530		US 20020064843 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
102	20020509		US 20020055160 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
103	20020321		US 20020034803 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
104	20020228		US 20020025570 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
105	20020207		US 20020015987 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
106	20011220		US 20010053844 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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107	20011213		US 20010051360 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
108	20040504		US 6730506 B2	Isolated human kinase proteins
109	20040406		US 6716604 B2	Nucleic acid molecules encoding a subunit of a human calcium/calmodulin-dependent protein kinase
110	20040316		US 6706511 B2	Isolated human kinase proteins
111	20040316		US 6706510 B2	Isolated human kinase proteins
112	20040217		US 6692948 B2	Isolated human kinase proteins
113	20040217		US 6692939 B2	Nucleotide sequences encoding mammalian calcium activated chloride channel-adhesion molecules

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114	20040210		US 6689597 B2	Isolated human kinase proteins
115	20040203		US 6686187 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
116	20040203		US 6686176 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
117	20040120		US 6680188 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
118	20031230	64	US 6670164 B2	Isolated human kinase proteins
119	20031230		US 6670163 B2	Isolated human kinase proteins
120	20031230		US 6670162 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
121	20031216		US 6664087 B2	Isolated human kinase proteins

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122	20031216		US 6664085 B2	Isolated human calcium/calmodulin (CaMk) dependent kinase proteins
123	20031125		US 6653117 B2	Isolated human kinase proteins
124	20031125		US 6653116 B2	Isolated human kinase proteins
125	20031118		US 6649389 B2	Isolated human kinase proteins
126	20031028		US 6638745 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
127	20031007		US 6630337 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
128	20031007		US 6630336 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
129	20031007		US 6630334 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
130	20030902		US 6613563 B1	Viral vectors with modified tropism

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131	20030624		US 6582946 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
132	20030617		US 6579709 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
133	20030520		US 6566130 B1	Androgen-regulated gene expressed in prostate tissue
134	20030429		US 6555352 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
135	20030422		US 6551795 B1	Nucleic acid and amino acid sequences relating to pseudomonas aeruginosa for diagnostics and therapeutics
136	20030325		US 6537788 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
137	20030318		US 6534299 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
138	20030304	86	US 6528294 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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139	20030211	716	US 6518013 B1	Methods for the inhibition of epstein-barr virus transmission employing anti-viral peptides capable of abrogating viral fusion and transmission
140	20030107		US 6503886 B1	Compositions containing nucleic acids and ligands for therapeutic treatment
141	20021231	65	US 6500938 B1	Composition for the detection of signaling pathway gene expression
142	20021231		US 6500656 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
143	20021231		US 6500655 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
144	20021210		US 6492156 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
145	20021210		US 6492155 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
146	20021210		US 6492154 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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147	20021210		US 6492153 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
148	20021203		US 6489153 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
149	20021119		US 6482935 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
150	20021119	67	US 6482624 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
151	20021112		US 6479269 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
152	20021112	747	US 6479055 B1	Methods for inhibition of membrane fusion-associated events, including respiratory syncytial virus transmission
153	20021105		US 6476195 B1	Secreted protein HNFGF20
154	20021008		US 6461846 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
155	20021001		US 6458561 B1	Human NIM1 kinase

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156	20020924		US 6455291 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
157	20020910		US 6448057 B1	Isolated human casein kinase proteins, nucleic acid molecules encoding human casein kinase proteins, and uses thereof
158	20020827		US 6441156 B1	Calcium channel compositions and methods of use thereof
159	20020820		US 6437110 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
160	20020730		US 6426206 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
161	20020723		US 6423521 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
162	20020709		US 6416990 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
163	20020702		US 6413756 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
164	20020625		US 6410294 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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165	20020611		US 6403353 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
166	20020514		US 6387677 B1	Nucleic acid molecules encoding human calcium/calmodulin (CaMK) dependent kinase proteins
167	20020416	87	US 6372468 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
168	20020122		US 6340584 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
169	20020122		US 6340583 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
170	20020101	227	US 6335170 B1	Gene expression in bladder tumors
171	20011218		US 6331423 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
172	20011127		US 6323016 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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173	20011030		US 6309857 B1	Nucleotide sequences encoding mammalian calcium activated chloride channel-adhesion molecules
174	20010508	723	US 6228983 B1	Human respiratory syncytial virus peptides with antifusogenic and antiviral activities
175	20010327		US 6207148 B1	Disease associated protein kinases
176	20000725	641	US 6093794 A	Isolated peptides derived from the Epstein-Barr virus containing fusion inhibitory domains
177	20000530	711	US 6068973 A	Methods for inhibition of membrane fusion-associated events, including influenza virus
178	20000509	641	US 6060065 A	Compositions for inhibition of membrane fusion-associated events, including influenza virus transmission
179	20000425		US 6054265 A	Screening assays for compounds that inhibit membrane fusion-associated events

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180	20000314		US 6037329 A	Compositions containing nucleic acids and ligands for therapeutic treatment
181	20000125		US 6017536 A	Simian immunodeficiency virus peptides with antifusogenic and antiviral activities
182	20000111		US 6013263 A	Measles virus peptides with antifusogenic and antiviral activities
183	19991005		US 5962232 A	Protein kinase molecules
184	19990323		US 5885803 A	Disease associated protein kinases

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1	20040429	48	US 20040081999 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
2	20040422	108	US 20040077048 A1	Protein modification and maintenance molecules
3	20040422	151	US 20040077044 A1	Kinases and phosphatases
4	20040408	53	US 20040067568 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
5	20040408	47	US 20040067522 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
6	20040401	68	US 20040063142 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
7	20040401	53	US 20040063130 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
8	20040318	144	US 20040053394 A1	Human kinases
9	20040304	397	US 20040043930 A1	Novel proteins and nucleic acids encoding same

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10	20040304	184	US 20040043466 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
11	20040226	152	US 20040038881 A1	Human kinases
12	20040226	52	US 20040038363 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
13	20040226	40	US 20040038362 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
14	20040205	144	US 20040023242 A1	Human kinases
15	20040129	112	US 20040018185 A1	Human kinases
16	20040122	53	US 20040014659 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
17	20040122	74	US 20040014193 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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18	20031218	111	US 20030232408 A1	ISOLATED HUMAN KINASE PROTEINS
19	20031211	40	US 20030228674 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
20	20031211	122	US 20030228595 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
21	20031113	136	US 20030211093 A1	Human kinases
22	20031113		US 20030211040 A1	Phosphodiesterase activity and regulation of phosphodiesterase 1B-mediated signaling in brain
23	20031106	128	US 20030207311 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
24	20031106	148	US 20030207299 A1	Human kinases
25	20030925		US 20030180786 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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26	20030918		US 20030175927 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
27	20030918		US 20030175926 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
28	20030918		US 20030175791 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
29	20030904		US 20030166221 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
30	20030904		US 20030166219 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
31	20030904		US 20030166218 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
32	20030904	85	US 20030166215 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
33	20030904		US 20030165891 A1	Novel TWIK-6, TWIK-7, IC23927, TWIK-8, IC47611, IC47615, HNMDA-1, TWIK-9 alpha2delta-4, 54414, and 53763 molecules and uses therefor

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34	20030821		US 20030157679 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
35	20030731		US 20030143690 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
36	20030724		US 20030140354 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
37	20030717		US 20030134319 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
38	20030710		US 20030129704 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
39	20030710	90	US 20030129645 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
40	20030626		US 20030119037 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
41	20030508		US 20030087294 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
42	20030424		US 20030077799 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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43	20030403	68	US 20030064475 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
44	20030320		US 20030054529 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
45	20030313		US 20030050230 A1	STE20-RELATED PROTEIN KINASES
46	20030313		US 20030049795 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
47	20030313		US 20030049792 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
48	20030220		US 20030036181 A1	Peptide extended glycosylated polypeptides
49	20030206		US 20030027307 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
50	20030130		US 20030022341 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
51	20030130		US 20030022340 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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52	20030130		US 20030022339 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
53	20030130		US 20030022337 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
54	20030130		US 20030022232 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
55	20030130		US 20030022229 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
56	20030102		US 20030003560 A1	Isolated human casein kinase proteins, nucleic acid molecules encoding human casein kinase proteins, and uses thereof
57	20021114		US 20020168741 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
58	20021017		US 20020151020 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
59	20021003		US 20020142430 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
60	20021003		US 20020142427 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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61	20020926		US 20020137167 A1	ISOLATED HUMAN CASEIN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN CASEIN KINASE PROTEINS, AND USES THEREOF
62	20020919		US 20020132325 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
63	20020919		US 20020132324 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
64	20020919		US 20020132322 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
65	20020912		US 20020127683 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
66	20020905		US 20020123121 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
67	20020905		US 20020123120 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
68	20020829		US 20020119548 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
69	20020829		US 20020119544 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF

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70	20020815		US 20020110889 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
71	20020815		US 20020110888 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
72	20020801		US 20020103116 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
73	20020718		US 20020094946 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
74	20020718		US 20020094560 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
75	20020718		US 20020094558 A1	Family of mammalian potassium channels, their cloning and their use, especially for the screening of drugs
76	20020704		US 20020086391 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEROF
77	20020627		US 20020082189 A1	ISOLATED HUMAN SERINE/THREONINE KINASE NUCLEIC ACID MOLECULES ENCODING HUMAN SERINE/THREONINE KINASE AND USES THEREOF

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78	20020620		US 20020076783 A1	Plants and plants cells expressing histidine tagged intimin
79	20020613	68	US 20020072491 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
80	20020530		US 20020064851 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
81	20020530		US 20020064843 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
82	20020509		US 20020055160 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
83	20020321		US 20020034803 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
84	20020314		US 20020032322 A1	Family of mammalian potassium channels, their cloning and their use, especially for the screening of drugs
85	20020228		US 20020025570 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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86	20020207		US 20020015987 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
87	20011220		US 20010053844 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
88	20011213		US 20010051360 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
89	20040504		US 6730506 B2	Isolated human kinase proteins
90	20040406		US 6716604 B2	Nucleic acid molecules encoding a subunit of a human calcium/calmodulin-dependent protein kinase
91	20040316		US 6706511 B2	Isolated human kinase proteins
92	20040316		US 6706510 B2	Isolated human kinase proteins
93	20040217		US 6692948 B2	Isolated human kinase proteins

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95	20040203		US 6686187 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
96	20040203		US 6686176 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
97	20040120		US 6680188 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
98	20040120		US 6680170 B2	Polynucleotides encoding STE20-related protein kinases and methods of use
99	20031230	64	US 6670164 B2	Isolated human kinase proteins
100	20031230		US 6670163 B2	Isolated human kinase proteins
101	20031230		US 6670162 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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104	20031202		US 6656716 B1	Polypeptide fragments of human PAK5 protein kinase
105	20031125		US 6653117 B2	Isolated human kinase proteins
106	20031125		US 6653116 B2	Isolated human kinase proteins
107	20031118		US 6649389 B2	Isolated human kinase proteins
108	20031028		US 6638745 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
109	20031007		US 6630337 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
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113	20030617		US 6579709 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
114	20030429		US 6555352 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
115	20030325		US 6537788 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
116	20030318		US 6534299 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
117	20030304	86	US 6528294 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
118	20021231		US 6500656 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
119	20021231		US 6500655 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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121	20021210		US 6492155 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
122	20021210		US 6492154 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
123	20021210		US 6492153 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
124	20021203		US 6489153 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
125	20021119		US 6482935 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
126	20021119	67	US 6482624 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
127	20021112		US 6479269 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
128	20021008		US 6461846 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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130	20020910		US 6448057 B1	Isolated human casein kinase proteins, nucleic acid molecules encoding human casein kinase proteins, and uses thereof
131	20020820		US 6437110 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
132	20020730		US 6426206 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
133	20020723		US 6423521 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
134	20020709		US 6416990 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
135	20020702		US 6413756 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
136	20020625		US 6410294 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
137	20020611		US 6403353 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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139	20020416	87	US 6372468 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
140	20020122		US 6340584 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
141	20020122		US 6340583 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
142	20011218		US 6331423 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
143	20011127		US 6323016 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
144	20011030		US 6309855 B1	Family of mammalian potassium channels, their cloning and their use, especially for the screening of drugs
145	20001024		US 6136581 A	Kinase genes and uses

	L #	Hits	Search Text
1	L1	304946	calcium or calmodulin
2	L2	323635	l1 (3w) kinase$2
3	L3	621609	clon$3 or express$3 or recombinant
4	L4	43722	l2 same l3
5	L5	1512978	human (w)l2
6	L6	610	human adj l2
7	L7	1991162	s l3 and l6
8	L8	602	l3 and l6
9	L9	113912	brain or lung or hippcampus
10	L10	526	l8 and l9
11	L11	0	"calcium/calmodulin protein kinase$2"
12	L12	0	"CaM kinase$2"
13	L13	0	"calmodulin binding protein kinase$2"

	L #	Hits	Search Text
14	L14	3838	"creb" or "CFTR" or "synapsin$3"
15	L15	184	l10 and l14
16	L16	12581	YAN KETCHUM MERKULOV BEASLEY
17	L17	146	l6 and l16
18	L18	146	l8 and l16
19	L19	145	l10 and l16

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1	20040429	48	US 20040081999 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
2	20040422	108	US 20040077048 A1	Protein modification and maintenance molecules
3	20040422	151	US 20040077044 A1	Kinases and phosphatases
4	20040408	53	US 20040067568 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
5	20040408	47	US 20040067522 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
6	20040401	68	US 20040063142 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
7	20040401	53	US 20040063130 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
8	20040318	144	US 20040053394 A1	Human kinases
9	20040304	397	US 20040043930 A1	Novel proteins and nucleic acids encoding same

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10	20040304	184	US 20040043466 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
11	20040226	152	US 20040038881 A1	Human kinases
12	20040226	52	US 20040038363 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
13	20040226	40	US 20040038362 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
14	20040205	144	US 20040023242 A1	Human kinases
15	20040129	112	US 20040018185 A1	Human kinases
16	20040122	53	US 20040014659 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
17	20040122	74	US 20040014193 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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18	20031218	111	US 20030232408 A1	ISOLATED HUMAN KINASE PROTEINS
19	20031211	40	US 20030228674 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
20	20031211	122	US 20030228595 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
21	20031113	136	US 20030211093 A1	Human kinases
22	20031113	50	US 20030211040 A1	Phosphodiesterase activity and regulation of phosphodiesterase 1B-mediated signaling in brain
23	20031106	128	US 20030207311 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
24	20031106	148	US 20030207299 A1	Human kinases
25	20030925	70	US 20030180786 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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26	20030918	102	US 20030175927 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
27	20030918	45	US 20030175926 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
28	20030918	210	US 20030175791 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
29	20030904	48	US 20030166221 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
30	20030904	79	US 20030166219 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
31	20030904	42	US 20030166218 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
32	20030904	85	US 20030166215 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
33	20030904	638	US 20030165891 A1	Novel TWIK-6, TWIK-7, IC23927, TWIK-8, IC47611, IC47615, HNMDA-1, TWIK-9 alpha2delta-4, 54414, and 53763 molecules and uses therefor

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34	20030821	41	US 20030157679 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
35	20030731	44	US 20030143690 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
36	20030724	61	US 20030140354 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
37	20030717	53	US 20030134319 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
38	20030710	76	US 20030129704 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
39	20030710	90	US 20030129645 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
40	20030626	156	US 20030119037 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
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43	20030403	68	US 20030064475 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
44	20030320	90	US 20030054529 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
45	20030313	222	US 20030050230 A1	STE20-RELATED PROTEIN KINASES
46	20030313	81	US 20030049795 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
47	20030313	47	US 20030049792 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins and uses thereof
48	20030220	74	US 20030036181 A1	Peptide extended glycosylated polypeptides
49	20030206	185	US 20030027307 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
50	20030130	89	US 20030022341 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
51	20030130	207	US 20030022340 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof

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52	20030130		US 20030022339 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
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54	20030130		US 20030022232 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
55	20030130		US 20030022229 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
56	20030102		US 20030003560 A1	Isolated human casein kinase proteins, nucleic acid molecules encoding human casein kinase proteins, and uses thereof
57	20021114		US 20020168741 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
58	20021017		US 20020151020 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
59	20021003		US 20020142430 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
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62	20020919		US 20020132325 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
63	20020919		US 20020132324 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
64	20020919		US 20020132322 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
65	20020912		US 20020127683 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
66	20020905		US 20020123121 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
67	20020905		US 20020123120 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
68	20020829		US 20020119548 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
69	20020829		US 20020119544 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF

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71	20020815		US 20020110888 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
72	20020801		US 20020103116 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
73	20020718		US 20020094946 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
74	20020718		US 20020094560 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
75	20020718		US 20020094558 A1	Family of mammalian potassium channels, their cloning and their use, especially for the screening of drugs
76	20020704		US 20020086391 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEROF
77	20020627		US 20020082189 A1	ISOLATED HUMAN SERINE/THREONINE KINASE NUCLEIC ACID MOLECULES ENCODING HUMAN SERINE/THREONINE KINASE AND USES THEREOF

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79	20020613	68	US 20020072491 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
80	20020530		US 20020064851 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
81	20020530		US 20020064843 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
82	20020509		US 20020055160 A1	ISOLATED HUMAN KINASE PROTEINS, NUCLEIC ACID MOLECULES ENCODING HUMAN KINASE PROTEINS, AND USES THEREOF
83	20020321		US 20020034803 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
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88	20011213		US 20010051360 A1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
89	20040504		US 6730506 B2	Isolated human kinase proteins
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105	20031125		US 6653117 B2	Isolated human kinase proteins
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107	20031118		US 6649389 B2	Isolated human kinase proteins
108	20031028		US 6638745 B1	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
109	20031007		US 6630337 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
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126	20021119	67	US 6482624 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
127	20021112	202	US 6479269 B2	Isolated human kinase proteins, nucleic acid molecules encoding human kinase proteins, and uses thereof
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